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OF THE

CTICE OF MEDICINE.

EDITED BY DR. H. VON ZIEMSSEN,
PROFESSOR OF CLINICAL MEDICINE IN MUNICH, BAVARIA.

VOL. XIV

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ASES OF THE NERVOUS SYSTEM,

AND

DISTURBANCES OF SPEECH.

BY

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BIOGRAPHICAL SKETCHES OF THE AUTHORS.

Adolph Kussmaul was born February 22, 1822, in the town of Graben, near Carlsruhe, Grand Duchy of Baden. His father, Jacob Philipp Kussmaul, city physician, died at Wiesloch in 1850; his mother, Louise Boehringer by name, was a native of Würtemberg. From his eighth to his tenth year he was under the instruction of an excellent country clergyman; subsequently he visited the gymnasia of Mannheim and Heidelberg. In the latter city he studied under Tiedemann, Gmelin, Henle, Puchelt, Pfeufer, Chelius, and Naegele, and passed his state examination in 1846. He was also an assistant under Naegele and Pfeufer. While a student he received a prize from the Medical Faculty of Heidelberg, the title of his essay being "The Color Phenomena at the Fundus of the Human Eye." This production, which was printed in 1845, exhibited the first effort—an abortive one, it is true—towards the construction of an ophthalmoscope.

During 1847, and part of 1848, Kussmaul was at Vienna and Prague, studying under Skoda, Rokitansky, Hebra, Oppolzer, Pitha, Dittrich, and others. In 1848 and 1849 he took part in the Holstein campaign as a volunteer. In 1850 he engaged in the practice of medicine at Kandern, in the Black Forest, Baden, but the severe taxations of a very large and onerous practice were too exhaustive, and he broke down in the spring of 1853. For four months he was paralyzed in both extremities, the result of spinal meningitis. Recovering from the attack in the following summer, he resolved to attempt the career of a university instructor. After renewing his studies for a year, Virchow took him to Würzburg, where he remained until the autumn of 1854. He then spent three months at the Lunatic Asylum of Illenau, but finally took up his residence in Heidelberg, where he became a private instructor in the summer of 1855, and, in 1857, Extraordinary Professor. While at Heidelberg, Kussmaul published numerous experimental articles in Moleschott's Untersuchungen and Virchow's Archiv, as also in the Prager Vierteljahresschrift; some articles were also published independently.

Of these we may mention "Researches on the Influence which the Circulation exercises upon the Motions of the Iris and other Parts of the Head," Verhandlun-

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gen der Würzburger medicinische Gesellschaft, 1856, Band 6. "On Rigor Mortis, etc.," Prager Vierteljahresschrift, Band 50, 1856. "Researches on the Origin and Nature of Epileptic Spasms and Epilepsy in General," Frankfurt, 1857 (published in conjunction with his friend A. Tenner). "On the Rupture of the Inner Membranes of the Cervical Arteries in Death by Hanging," Virchow's Archiv, 1857. "On the Death of Limbs after Injections of Chloroform into the Arteries," Virchow's Archiv, 1858. "On the Absence, Malformation, and Doubling of the Uterus; Misconception and the Migration of the Ovum," Würzburg, 1859. In the autumn of 1859 Kussmaul removed to Erlangen, where he became Professor of Internal Medicine and Director of the Medical Clinic and Polyclinic, remaining in this city until the spring of 1863.

Of numerous dissertations which have appeared under his authority, such as that of Max Doederlein, "On the Diagnosis of Cancerous Tumors in the Right Hypochondrium," 1860, which perhaps deserves special notice, there are also: "Researches on Constitutional Mercurialism," Würzburg, 1861. "On Sexual Maturity," Würzburger medicinische Zeitschrift, 1862. "Contributions to the Anatomy and Pathology of the Urinary Apparatus," Würzburger medicinische Zeitschrift, 1863. "A Free Concretion in the Pericardium," *Ibid.*, 1864. In the spring of 1863 he accepted a call to Freiburg in Breisgau. Here he received calls to Basel, Königsberg, Greifswald, Tübingen, and Breslau, all of which he refused. In Freiburg he published numerous articles. Of these we may mention the following: "On the Diagnosis of Embolism of the Mesenteric Artery," Würzburger med. Zeit., 1864. "The Cold in the Head of Infants," Henle and Pfeufer's Zeitschrift f. rationelle Medicin, 1865. "On Congenital Narrowing and Closure of the Pulmonary Arteries," *Ibid.*, 1865. "The Developmental Periods of Exact Medicine." "On the Causes and the Phenomena of our Death" (two public addresses), Freiburg, 1865. "On Periarteritis Nodosa" (in conjunction with Prof. R. Maier), Deutsches Archiv, 1866. "The Lungs and Bronchial Glands after Incineration," after analyses by Dr. C. W. Schmidt, *Ibid.*, 1866. "On Poisoning by the Chloride of Zinc" (in conjunction with Dr. Honsell), Klinische Wochenschrift, 1866. "Several Observations on Rare Affections of the Liver," *Ibid.*, 1867 and 1868. "Sixteen Observations on Thoracentesis in Pleurisy, Empyema, and Pyopneumothorax," Deutsches Archiv f. klin. Med., 1868. "On the Treatment of Gastric Dilatations, by a New Method—the Stomach-Pump," *Ibid.*, 1869. "Twenty Letters on the Inoculation of Variola and Vaccinia—A General Exhibit of the Vaccination Question," Freiburg, 1870. "On Rheumatic Tetanus," Berl. klin. Woch., 1871. "On the Pathological Anatomy of Chronic Saturnismus" (with R. Maier), Deutsches Archiv, 1872. "On the Doctrine of Tetanus," Berl. klin. Woch., 1872. "On an Abortive Form of Tetanus," Deutsches Archiv f. klin. Med., 1873. "On Spontaneous Gradual Closure of the Trunk of the Great Vessels of the Neck," Deutsche Klinik, 1872. "On Progressive Bulbar Paralysis," Sammlung klinischer Vorträge, by Volkmann, 1873, No. 54.

In the autumn of 1876 he went to Strassburg, to take the direction of the Clinic and Polyclinic. Professor Kussmaul married Louise Wolf, in 1850. One of his daughters is the wife of Professor Czerny, who was recently called from Freiburg to Heidelberg, as the successor of the deceased Simon.

ALBERT EULENBURG was born August 10, 1840, in Berlin. His father was the well-known orthopædist, who first introduced the Swedish movement-cure into Germany, and utilized it in a scientific manner. His studies were carried on in Bonn and Berlin, and in the year 1861 he took a prize on the following question proposed by the Berlin University: "On the Influence of Cardiac Hypertrophy and Disease of the Cerebral Arteries on the Occurrence of Cerebral Hemorrhage," Virchow's Archiv, Band XXIV. His graduation dissertation of the same year was "De argumentis irritabilitatis muscularis recentioribus." In 1863 he became Assistant Physician at the University Hospital in Greifswald; in 1864 he took the prize founded by Hufeland's Society, for the best work on the hypodermic injection of remedies, printed in 1865, under the title "The Hypodermic Injection of Remedies —after Physiological Researches and Clinical Experiences," August Hirschwald, second edition, 1869; third edition, 1875. In 1866 he established himself as a private instructor at the University of Berlin. From 1869 to 1871 he was Assistant at the Polyclinic of the University. He took part in the campaigns of 1864, 1866, and 1870 to 1871; in the latter year he published his "Manual of Functional Nervous Diseases," August Hirschwald, Berlin; and in 1873, in conjunction with Dr. Paul Guttmann, his "Pathology of the Sympathetic." Since 1874 he has been Professor of Medicine at the University of Greifswald. Eulenburg has also published numerous small articles in the domain of neural pathology and electrotherapy, as also in experimental pharmacology (on quinine, narcein, bromine, calcium, transfusion, acute poisoning by phosphorus, nitrite of amyl, etc.), most of which have appeared in the Archives of Anatomy and Physiology and Pathological Anatomy, and in the German Archiv für klinische Medicin. Eulenburg is an honorary member of the New York Neurological Society, and corresponding member of the New York Society of Neurology and Electrology.

FREDERICK JOLLY was born in Heidelberg, November 24, 1844, but in 1854 his family moved to Munich, where he attended the gymnasium, and in the autumn of 1862 the university. He studied medicine in Munich and Goettingen, and in the former city he was for two years Assistant at Pfeufer's Medical Clinic. While in Berlin and Vienna he turned his attention chiefly to mental diseases, then entered the Bavarian Lunatic Asylum of Werneck, as Assistant, later accepting a similar position at the insane department of the Julius Hospital in Würzburg. There, in 1871, he was installed as private instructor, and in 1873 responded to a call as Extraordinary Professor of Psychiatry and Director of the Psychiatric Clinic at the University of Strassburg. In the beginning of the year 1875 he was made full professor. His literary productions relate in part to the domain of the anatomy and physiology of the nervous system, in part to questions of psychiatry and neural pathology. They are: "On the Ganglion-Cells of the Spinal Cord," Zeitsch. f. wiss. Zoologie, XVII. "A Case of Faulty Development of the Bulbus in the Human (Male) Brain," Zeitschr. f. rat. Med., 3, XXXVI.

"On Traumatic Encephalitis," in Stricker's Studien aus dem Institute f. experimentelle Pathologie, 1869. "Researches on Cerebral Pressure and on the Blood-Movements in the Skull," Würzburg, 1871.

"Report on the Insane Department of the Julius Hospital," Würzburg, 1873. The Archiv f. Psychiatrie also contains some articles by him, on "Psychoses in Consequence of the Campaign of 1870 and 1871," on "Multiple Cerebral Sclerosis," on "The Electric Examination of those having Hallucinations of Hearing," "On the Care of the Insane at Home in Scotland." Various other articles have also appeared in the medical weeklies.

Herrmann Senator was born December 6, 1834, in Gnesen, where he pursued his early studies. From 1845 to 1853 he attended the Frederick William Gymnasium in Posen, and then the University of Berlin, where he graduated in 1857. On March 1st of the following year he received his medical degree and established himself in Berlin. During his period of study he was for a year and a half an amanuensis under Johannes Mueller, and as such he was chiefly concerned with anatomy and physiology.

From 1860 onward he devoted himself to state medicine, and later mostly to general pathology and medical chemistry in Virchow's Pathological Institute (under the instruction of W. Kuehne). In August, 1868, he established himself as instructor, and from 1869 to 1872 pursued the study of clinical medicine at the clinic of Traube.

In March, 1875, he received charge of the inner department of the Augusta Hospital, and the polyclinic attached to it, and in September of the same year he was made Extraordinary Professor of the Medical Faculty.

Eugene Seitz was born at Vilbel, in the Grand Duchy of Hesse, and since the year 1856 has held the position of Professor of Special Pathology and Therapeutics at Giessen, having at the same time the management of the medical clinic. While assistant and private instructor in Giessen and Tübingen, it was his intention to become an oculist, and he commenced a manual of ophthalmology, but relinquished it to Professor Zehender when his attention was turned towards general medicine. His principal work has been the complete recasting of Niemeyer's Manual of Special Pathology, which, at the death of the latter, was placed in his hands. He also wrote a text-book on Percussion and Auscultation of the Respiratory Organs, which was published at Erlangen in 1866. Among the smaller productions of his pen, and the numerous inaugural dissertations prepared under his directions, the following are the most important: "Percussion of the Upper Borders of the Lungs" (Diss. Heyer). "On the Changes in the Lungs during Disease" (Diss. Neidhart). "On Changing Respiratory Murmurs," Deutsche Arch. f. klin. Med. "Researches on the Arterial Pulse by Means of Marey's Sphygmograph" (Diss. Arcularius). "Percussion of the Spleen" (Diss. Schüster). "Situs viscerum inversus" (Diss. Steinhauser). "Simple Scleroma of the Skin" (Diss. Nordt), etc.

Carl Ferdinand Hermann Immermann was born in Magdeburg on September 2, 1838. After completing his courses at the schools of his native city, he entered the university in the autumn of 1856. From this year on until 1861 he attended the high schools of Halle, Würzburg, Greifswald, Tübingen, and Berlin, and in the latter city obtained the degree of Doctor in Medicine. In 1861, the following year, while in Berlin, he also passed the Prussian state medical examination. During 1862 and 1863 he continued his studies in Berlin, Vienna, and Paris, making it his special object to attend clinics and courses, in preparation for his intended career in the field of clinical medicine. In Easter, 1864, he visited Tübingen, to take the position of Assistant at the Medical Clinic of Felix von Niemeyer, whose clinical instruction he had followed with particular zest as a student, and whose very warm interest and attention he had always enjoyed. Though he did not establish himself formally as an instructor, he still gave practical courses in clinical diagnosis; but, after remaining two years, at the solicitation of Professor von Ziemssen he moved to Erlangen and received from him the position (which had just become vacant) of Assistant at the Medical Polyclinic. In the summer of 1866 he established himself as private instructor. During the following two years he continued his service as Assistant at the Polyclinic and Instructor, while he delivered lectures on separate branches of Special Pathology, such as Diseases of the Skin and Syphilis, on Materia Medica and the Art of Prescribing, and continued to hold courses in clinical diagnosis as before in Tübingen. Finally, in the autumn of 1871, he responded to a call from Basel, and since that time has been serving as Regular Professor of Special Pathology and Therapeutics, Director of the Medical Clinic, and Physician in Chief at the City Municipal Hospital. His literary productions are: "De morbis febrilibus questiones nonnullæ, adjectis morborum historicis," Diss. Inaug., Berlin, 1860. "Contributive Cases to the Theory of the Rise in the Temperature during Fever," Deutsche Klinik, 1865, Nos. 1–4. "On Vertigo in Diseases of the Posterior Fossa Cranii, and their Connection with Actual Oscillation of the Body," Deutsches Archiv f. klin. Med., Bd. I., S. 595 ff. From von Niemeyer's clinic, "Four Cases of Cerebral Disease," Berl. klin. Woch., 1865, p. 177. "On the Pathogenesis and Etiology of the Visible Respiratory Swelling of the Cervical Veins," Habilitation essay, Erlangen, 1866. "Stricture of Both Principal Trunks of the Pulmonary Artery through Chronic Interstitial Pneumonia," Deutsches Archiv f. klin. Med., V., p. 235. "Pneumonia and Meningitis," *Ibid.*, V., p. 1 (in connection with A. Heller). "The Cold-Water Treatment of Typhoid, after Observations at the Erlangen Clinic and Polyclinic," Leipsic, 1870 (in connection with H. von Ziemssen). "On the Therapeutics of the Diurnal Variations of Temperature in Typhoid Fever," Deutsches Archiv f. klin. Med., VII., p. 561. "On the Therapeutic Aims of Internal Medicine at the Present Time". opening address in assuming charge of the Medical Clinic in Basel, Leipsic, 1871. "On Morbus Brightii and its Treatment," Correspondenzblatt f. Schweizer Aertze, 1873, No. 11. "On Progressive Pernicious Anæmia," Deutsches Archiv f. klin. Med., XIII., p. 209.

Dr. F. V. Birch-Hirschfeld was born May 2, 1842, near Rendsburg, on the

Manor Cluvensieck, in Holstein. His grandfather on the mother's side, an Englishman, having daughters, but no sons, was very desirous that his name should be preserved by the children, whence the double name. The German portion belongs to an old Thuringian Lutheran family, which can be traced back into the sixteenth century.

His school education was obtained at the gymnasia of Kiel and Hamburg, and in the year 1862 he matriculated as a student of medicine in Leipsic. After nearly five years of study, broken in 1866 by long active service in hospital, he was made assistant to Professor E. Wagner at the University Polyclinic and then at the Pathological Institute. After several years of activity in these positions he entered upon the career of a government physician, for a year and a half studying mental diseases and acting as physician of the provincial institutions Sonnenstein and Colditz in Saxony. In the year 1870 he received the position of prosector at the Dresden City Hospital, and was soon after made instructor in pathological anatomy in the educational course of the Sanitary Corps of the Royal Saxon Army, and later was made circuit physician of the Provincial Blind Asylum. Of his more important scientific works the following are to be mentioned: 1. On a Case of Cerebral Deficiency in consequence of Dropsy of the Septum Lucidum, Diss. Leipsic, 1867 (published in the Archiv f. Heilkunde, 1867). 2. The Development of Cancer of the Testicle, Archiv der Heilkunde, 1868. 3. Cases of Embolism in Tumors, Archiv der Heilkunde, 1869. 4. On Miliary Tuberculosis after Abdominal Typhus, Arch. der Heilkunde, 1870. 5. On the Question of Cylindroma, Arch. der Heilkunde, 1870. 6. Acute Splenic Tumor, *Ibid.*, 1872. 7. Investigations on Pyæmia, *Ibid.*, 1873. 8. Transfusion of Lamb's Blood (in conjunction with Dr. A. Fiedler), Deutsches Archiv f. klin Med., XIII. 9. Contributions to the Pathological Anatomy of the Hereditary Syphilis of New-born Children, Archiv f. Heilkunde, 1874. 10. Manual of Pathological Anatomy, Leipsic, 1876–7 (Vogel).

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(Translated by E. Buchanan Baxter, M.D.)

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(Translated by George B. Shattuck, M.D.)

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(Translated by John A. McCreery, M.D.)

VASO-MOTOR AND TROPHIC NEUROSES.

EULENBURG.

HEMICRANIA.

(Migraine.)

Wepfer, Observat. med. pract. de affect. cap.—*Fordyce*, Historia febris miliaris et de hemicrania dissertatio. London, 1758.—*Tissot*, Traité des nerfs et de leurs maladies. Paris, 1783. T. III. 2.—*Schoenlein*, Allgemeine und specielle Therapie. 1832. IV.—*Andral*, Cours de pathologie interne (3. ed.). 1839.—*Valleix*, Traité des névralgies ou affections douloureuses des nerfs. 1841.—*Pelletan*, De la migraine, etc. Paris, 1843.—*Romberg*, Lehrbuch der Nervenkrankheiten. 2. Aufl. 1851. I.—*Leubuscher*, Krankheiten des Nervensystems. 1860.—*Du Bois-Reymond*, Zur Kenntniss der Hemikranie. Archiv für Anat. und Physiol. 1860. p. 461.—*Brown-Séquard*, De l'hémicranie, etc., in Journal de physiol. 1861.—*Lebert*, Handbuch der praktischen Medicin. 1862. II. 2.—*Eulenburg* and *Landois*, Die vasomotorischen Neurosen. Wiener med. Wochenschrift. 1867. No. 87.—*Frommhold*, Die Migraine und ihre Heilung durch Elektricität. Pest, 1868.—*Ferrand*, Union méd. 1868. 14.—*Moellendorf*, Ueber Hemikranie. Archiv für path. Anat. XLI. p. 385.—*Hasse*, Krankheiten des Nervensystems. 2. Aufl. 1869.—*Eulenburg*, Lehrbuch der functionellen Nervenkrankheiten. 1871.—*Anstie*, Neuralgia and the diseases that resemble it. London, 1871.—*Althann*, Beiträge zur Physiologie und Pathologie der Circulation. Dorpat, 1871.—*Brunner*, Zur Casuistik der Pathologie des Sympathicus. Petersb. med. Zeitschrift. N. F. II. 1871. p. 260.—*Berger*, Das Amylnitrit, ein neues Palliativmittel bei Hemikranie. Berl. klin. Wochenschrift. 1871. No. 2.—*Holst*, Ueber das Wesen der Hemikranie und ihre elektrotherapeutische Behandlung nach der polaren Methode. Dorpater med. Zeitschr. 1871. II. p. 261.—*Clifford Allbutt*, British Med. Journal. 1872. 10.—*Liveing*, On megrim, sick headache, and some allied disorders. London, 1873.—*Lasègue*, De la migraine, Arch. gén. Nov. 1873. p. 580.—*Berger*, Zur Pathogenese der Hemikranie. Virchow's Archiv. LIX. Heft 3 und 4. 1874. p. 315.—*E. Fraenkel*, Zur Pathologie des Hals-Sympathicus. Diss. Breslau, 1874.

By hemicrania we understand a series of symptoms, of which the most characteristic are *spontaneous attacks or fits of pain in* *the head, limited to one side, though often not distinctly bounded.* As a rule, the intervals between the attacks are quite considerable, and are free from pain. The disease is usually very chronic, tormenting the patient for many years, or his whole life, and is largely dependent upon congenital tendencies, or predisposition to neuropathy.

History.

The name "hemicrania" occurs in the old authors, but not always in a sense exactly corresponding to the description given by us. For instance, Bartholin[1] describes by the name of "hemicrania periodica" a case which seems in all likelihood to have been a typical supra-orbital neuralgia, occurring at fixed hours in the day. The older authors in general (Wepfer, Tissot, and others) confound the disease with supra-orbital neuralgia; even Schoenlein, who classes hemicrania among the neuroses of the genital system, and names it "hysteria cephalica," places the seat of pain in the ramifications of the frontal and temporal nerves. Among modern pathologists who adopt this position may be specially mentioned Lebert, Stokes, Anstie, Clifford Allbutt. Those who entertain these views have subdivided the disease in a somewhat arbitrary way, according to the presumed or actual, the predisposing or occasional, cause. Sauvages assumed ten such causes; Pelletan distinguished a "migraine stomacale, irienne, utérine, pléthorique;" Monneret and Fleury, a "migraine idiopathique" and "sympathique," in which division Valleix coincided, without giving a decided opinion regarding the localization of the disease. Piorry placed the seat of migraine in the nerves of the iris! An important step was taken by Romberg when he associated hemicrania with "hyperæsthesia of the brain," or cerebral pain, distinguishing it sharply from peripheral neuralgia, and plainly naming it "neuralgia cerebralis." He was followed by Leubuscher, among others, who calls hemicrania "true neuralgia of the brain." The negative merit of Romberg's view was certainly greater than the positive; a distinct demonstration of the cerebral origin of hemicrania was by no means given by Romberg and Leubuscher. The latest steps in the study of hemicrania are specially connected with the interesting observations of du Bois-Reymond, which led this celebrated physiologist to assume that certain forms of migraine were caused by a unilateral tetanus of the vessels of the head, or tetanus in the district supplied by the cervical sympathetic ("hemicrania sympathico-tonica"). Moellendorff, on the other hand, sought at a later time to show that hemicrania depended on unilateral relaxation of the vessels of the head, from want of energy of the vaso-motor nerves. For a number of years past I have taken a point of view between these two, and affirmed the partial correctness of both by attempting to show that a certain class of cases of migraine must be understood as vaso-motor in origin ("hemicrania vasomotoria"); but this class must be subdivided into the *sympathico-tonic* and the *angio-paralytic* or *neuro-paralytic* forms. This distinction, which is not without its value in therapeutics, seems to have met with rather general acceptance, as shown by later works (Brunner, Berger, Holst, and others).

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[1] Misc. curiosa sive Ephemerid. nat. curios. I. 1684. p. 130.

Etiology.

The etiology of hemicrania, as of so many other neuroses, is very little understood. In fact, we know only a number of what are called "predisposing circumstances" of so general a character that they claim almost equal importance in the causation of a number of other diseases of the nervous system; this very circumstance, however, is one which throws some light upon the family relation between hemicrania and the other neuroses of this group, and enables us to refer them all to one common basis, under the denomination of "constitutional neuropathies."

Among these predisposing circumstances we should attach very special importance to the influences of *sex*, *age*, and *hereditary tendency*.

1. *Sex.*—The female sex is very much more disposed to hemicrania than the male, the proportion being about that of 5 to 1. In the policlinic of Berlin, among the poor of the lower orders I

counted in fifteen months only two cases among men to thirteen in women. In private practice among the better classes the preponderance among women is equally evident. It is proper to remark that the predisposition to neuralgias in general is decidedly greater among women than among men; but by no means is this the case with the other neuralgias to the same extent as with hemicrania. Within the period of time above specified I counted thirty cases of superficial cutaneous neuralgia in men to seventy-six in women, a proportion of 2 to 5. The predisposition of women, however, to other forms of neuralgia about the head (those of the trigeminus and occipitalis) appears quite as decided as that to hemicrania. In my tables the proportion of cases in males and females respectively was, for trigeminus neuralgia, 5 to 24; for occipital neuralgia, 2 to 10. In neuralgias of the extremities a directly opposite ratio appeared, viz., brachial neuralgia, 4 to 2; lumbar neuralgia, 3 to 0; sciatica, 11 to 3. This predisposition of the female sex to hemicrania, and to neuralgias of the head in general (trigeminal and occipital), is certainly a circumstance deserving of attention. It points out to us the possible relation of these neuroses to normal or diseased menstruation, to the catastrophes and crises of the sexual life in women, to the pathological alienation of the entire nervous activity (hysteria) which is peculiar to women. Yet, on the other hand, it is beyond question that the male sex may, though more rarely, be attacked with neuralgia, and it follows that those authors have certainly gone too far, who (as for instance, Schoenlein) explain hemicrania as simply dependent upon diseases of the female generative system, and as a mere symptom of hysteria.

2. *Age.*—Youth is decidedly predisposed to the development (or, more correctly speaking, the outbreak) of migraine. The ingenious Tissot affirmed, albeit with some degree of exaggeration, that the person who should not be attacked by migraine before the twenty-fifth year of life would remain exempt therefrom for the rest of his days. The disease may occur during childhood, although at this period neuralgia is one of the rarest of all occurrences. The only cases of neuralgia that I have observed earlier than the tenth year were cases of hemicrania in girls, developed upon a decided hereditary basis (see further on).

The development of puberty very especially favors the outbreak of hemicrania; most hemicranias, whether hereditary or not, make their first appearance at this time. The period when hemicrania is most common is decidedly that between puberty and the fiftieth year of life, or thereabouts; it is certainly rare by comparison in later life, as some of the old cases get well, and new ones do not develop.

3. *Hereditary predisposition.*—The fact of inheritance is as well established in hemicrania, and its occurrence is almost as common, as in various other neuralgias and certain neuroses (epilepsy, paralyses, hysteria, insanity, etc.). The disease follows the female line, being usually inherited from the mother only, and by the daughters only. This is a natural inference from what has been said about sexual predisposition. If hereditary tendency is present, girls of even four or five years may be attacked by migraine, as I have repeatedly observed. In the case of a girl aged nine, who had had exquisite attacks of hemicrania from her fourth year upward, the mother had suffered from hemicrania from her earliest youth, and one sister was subject to epileptic attacks. This frequency of hereditary transmission, and the related fact of occurrence in childhood, compel us to include it with the neuroses above enumerated in the class of *constitutional neuropathies*, first distinctly formulated by Griesinger, the essential characteristic of which class must probably be stated as consisting in *congenital anomalies in the primary structural development of the central nervous apparatus.* It must, however, be admitted that no defensible and satisfactory statement of what these anomalies are has yet been made. Another circumstance adds weight to this view: it is extremely common to observe, not only a community in inheritance, but also a coincidence or alternation between hemicrania and certain other members of this group, especially epilepsy and insanity. As instances of this, it is sufficient to point out the fact that, in those epileptics who are predisposed by inheritance and constitution, attacks of migraine are among the commonest accompaniments of developed epilepsy, both in early and in later years; and that in families which have tendencies to constitutional neuropathic diseases there are often individual members who suffer with

migraine, while others are attacked with epilepsy, insanity, and other morbid conditions of this class.

The great probability of a constitutional neuropathic origin in many cases of migraine ought not to mislead us into a one-sided view, which excludes other causes, as has happened now and then in the case of other neuroses. The influence of heredity and constitutional tendency, however weighty, ought to be balanced in our minds by the equally important possibility of other factors, which may be termed, in a sense, accidental.

Compared with the predisposing influences of sex, age, and inheritance, the effects of such circumstances as dyscrasiæ, habits of life, social position, occupation, etc., are less easily demonstrated in hemicrania. The influence of certain dyscrasiæ in producing hemicrania cannot, at all events, be distinctly shown. Anæmic, chlorotic, syphilitic, arthritic persons, and those suffering from mercurial dyscrasia, are doubtless often attacked by hemicrania, but hardly oftener in proportion than other persons, unless we take the unjustifiable step of calling every symptomatic headache in such persons hemicrania, as has been done. Neither has hemicrania a special right to be regarded as a symptom of hysteria, although the headache of hysterical persons, called "clavus," has some resemblance to hemicrania. Still less justified are we in laying weight (as is often done) upon the influence of general or so-called abdominal plethora, or of sedentary, idle, or too opulent and luxurious habits. Hemicrania occurs in all professions and ranks of life: it is a disease of the poor day-laborer's wife as well as of the rich and *blasée* lady of fashion, although the former is not in a position to pay as much attention to her migraine and to make claims upon the attention of others on its account. Among men it affects the slender and weakly as well as the robust and those who bear the traces of indulgence in the pleasures of the table. While it is certainly obvious that learned men and those who work with their heads suffer relatively often from migraine, yet they owe this fatal favor not to their sedentary life, and still less to an indulgence in luxury, but rather to the concentrated tension of their mental activity, to the excess of functional stimulation of the brain, continuous or unnaturally increased from time to time.

Of the direct causes of hemicrania we know really nothing, and it is better to confess this ignorance at once than to make long détours in reaching a confession. That migraine stands in a certain etiological connection with local or general disturbances in the circulation of the blood, is a fact that did not escape the attention of older observers. The special frequency of attacks in women at the time of menstruation, and the identity in type with the latter phenomenon, as well as the improvement or disappearance of the disease after the climacteric period, must have called their attention to this point. The observations and conclusions recently published by du Bois-Reymond, Moellendorff, and others, respecting the mode in which local disturbances of circulation occur in many cases of migraine, have apparently brought the problem nearer to its solution; but, in truth, the result of this has only been the pointing out of the nervous paths which, by their periodic excess or depression of function, bring about the local disturbances of circulation which often accompany the attack of migraine. The causal relation of these local disturbances to the characteristic and cardinal phenomena of the attack of migraine is still in great need of an explanation, and the real etiology of migraine has, in point of fact, nothing to show, since the cause of the abnormal periodic excitement, or of the periodic variation in the excitability of the nerve-paths in question, is completely veiled in obscurity. (Cf. below, "Analysis of the Separate Symptoms.")

Pathology.

General Symptoms and Course.

The disease called hemicrania may be described as a succession of attacks, between which occur intervals of longer or shorter duration, usually free from symptoms. The separate attacks agree, on the whole, in certain cardinal symptoms, but are distinguished by considerable points of difference, sufficient to justify the establishment of distinct types of disease.

The attack of migraine is often preceded by certain prodromal symptoms. The patient feels on the day before, or in the forenoon of the day of the attack, a slight depression, a sense of pressure in the head, weariness, and indisposition to continued work. The attack is often also preceded by paræsthesiæ in the region of the higher organs of sense (muscæ volitantes, tinnitus aurium), as well as by a chill, morbid yawning, sneezing, or nausea. In some cases a violent enteralgia or gastralgia has been observed on the evening preceding each attack (Tissot, Berger) ; in other cases extreme hunger has been a prodromal symptom (Willis).

The patient often wakes with the characteristic *pain;* in other cases it comes on by degrees in the course of the day ; but it almost never comes with the lightning rapidity of neuralgic pain, as is the case, for example, in so many attacks of tic douloureux, which may also be evoked in its fullest severity by the most trifling external causes. The pain of hemicrania, as the name implies, is generally confined to one-half of the cranium, but not so strictly as that the limit of pain does not move back and forward across the median line. The left side is far oftener attacked than the right—in my observation, in the ratio of two to one. Then there are not a few persons who are attacked by turns on each side ; but in this case one side of the head is usually attacked oftener and more severely than the other. I have observed such cases—which may be designated as *hemicrania alternans*—in considerable numbers, and must make the remark that this special class presents in an exceptionally distinct manner the vaso-motor disturbances which are presently to be described.

The pain is, on the whole, not so much a variable or mobile as a fixed pain, however great may be its variations in intensity. It is not usually distributed uniformly over the whole side of the cranium, but is usually worse either on the fore part or else on the side or middle parts—the frontal, parietal, and temporal regions. The epithets which patients apply to it—dull, boring, bursting—are interesting, as differing from those we commonly hear in other neuralgias, especially in prosopalgia (as piercing, tearing, darting, etc.), and as agreeing more with the descriptions applied to clavus hystericus and cephalæa syphilitica.

Real *painful points*, in Valleix's sense, are entirely absent in pure hemicrania. The supra-orbital and temporal branches of the trigeminus are usually insensitive to pressure. The so-called *parietal point* is oftener found—a spot sensitive to pressure, somewhat above the tuber parietale, which has been referred, in rather a forced manner, sometimes to the recurrent branch of the trigeminus, sometimes to anastomoses of various cutaneous nerves (frontal, temporal, and occipital). Probably it is merely a case of *cutaneous hyperalgesia*, such as often exists in the attack of migraine, both in a circumscribed and a diffuse form. In many cases the greater part of the forehead, temples, and parietal region is sensitive to very light touches. On the other hand, a deep, diffused pressure upon these parts will often give some relief to the pain.

Besides these hyperalgesiæ of the affected side of the head, we find in many attacks of migraine, especially those which are connected with vaso-motor disturbances, that deep pressure gives decided pain when applied to *the region corresponding to the ganglion cervicale supremum of the cervical sympathetic*, or to the *ganglion cervicale medium;* sometimes also when applied to *the spinous processes of the lowest cervical and the first dorsal vertebræ.*

Besides these cutaneous hyperalgesiæ, there may exist a *pathological acuteness of the sense of touch* (*hyperpselaphesia*) in the affected side, as O. Berger[1] has lately shown by accurate tests of the sensibility in a case accompanied by fluxionary hyperæmia (hemicrania angio-paralytica). For instance, in corresponding parts of the frontal region he found the diameter of the circle of perception of touch to be on the right or affected side one line, while on the left side it was four lines. Variations of temperature of 0.4° C. were perceived on the right side, of 0.8° C. on the left; and, similarly, the electro-cutaneous test indicated sensibility at a minimum distance between the coils of 160 millim. on the right, 120 on the left.

The attack of hemicranial pain is often conjoined with nausea, vomiting, and the paralgiæ already mentioned in the region of the opticus and the acusticus, namely, muscæ volitantes, fiery circles before the eyes, tinnitus, etc. The unpleasant, foul taste, of which many patients complain before and during the attack, is probably a paralgia of the nerves of taste, and is not connected with gastric derangement, as is usually supposed.

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[1] Virchow's Archiv. LIX. Heft 3 and 4. p 324.

In a great number of cases the attack is accompanied by peculiar *local irregularities of circulation, temperature, and secretion*, and by peculiar phenomena in connection with the *eye*, which may be comprehended under the designation of *vaso-motor* and *oculo-pupillar symptoms*. According to the grouping and the coincidence in point of time of these phenomena, *two forms, in a sense typical*, can be distinguished, which are marked with perfect clearness in but a few cases, while in others they are only confused and indistinct, or irregularly mixed.

1. At the height of the attack the face is pale and sunken upon the side which is painful, the eye is sunken, the pupil dilated, and the temporal artery feels like a hard cord. The ear is also pale, and colder than the other ear; the temperature of the external meatus may fall from 0.4° to 0.6° C., according to my measurements. (I cannot regard as correct the much greater differences of temperature which some authors claim to have found.) The pain is aggravated by circumstances which increase the blood-pressure in the head, as stooping, coughing, etc., and increases synchronously with the pulsations of the carotid. I have observed that compression of the carotid upon the affected side may also increase the pain in such cases, while compression of the carotid on the well side gives relief. The salivary secretion may be abundant and viscid (O. Berger). Towards the end of the attack the pale part of the face and ear becomes reddened, with a sensation of heat and a rise in temperature, redness of the conjunctiva, lachrymation, and sometimes contraction of the pupil (previously dilated); the heart palpitates, the pulse is hastened, a general warmth is felt; there is abundant vomiting, a desire to make water, discharge of watery urine, and in some cases a diarrhœal watery stool. Attacks following this general course are called *hemicrania spastica or sympathico-tonica.*

2. At the height of the attack the affected side of the face is reddened, hot, and turgid, the conjunctiva brightly injected, the

secretion of tears increased, the pupil more or less closely contracted. Sometimes we find also a narrowing of the palpebral fissure, retraction of the globe, and a falling of the upper lid, with difficulty in performing its motions. The ear of the side attacked is also red and hot; the temperature of the outer meatus is raised—in my observations, from 0.2° to 0.4° C. The secretion of sweat is increased; sometimes there is ephidrosis unilateralis. The temporal artery is enlarged and beats with increased force; sometimes the carotid of the affected side does the same. Compression of the last-named vessel eases the pain, while compression of the other carotid makes it worse. The pulse may be retarded, beating from forty-eight to fifty-six times a minute (Moellendorff), while the radial artery is small and contracted; in some cases these phenomena are not present. The examination with the ophthalmoscope shows, in a few cases, dilatation of the arteria and vena centralis retinæ on the affected side, contortion of the vein, and dilatation of the vessels of the choroid, and a darkening of the tint of the fundus oculi (Moellendorff); but sometimes the condition is normal (Berger, upon the authority of H. Cohn's examination). Towards the end of the attack the reddened portions of the face become gradually paler, and the other phenomena pass off at the same time. For the attacks which follow this type I have proposed the name of *hemicrania angioparalytica* or *neuroparalytica.*

There seems to be a rare form of hemicrania, of which the single paroxysms present alternately the symptoms of the sympathico-tonic and of the neuroparalytic forms. A case of this sort has lately been described by Berger,[1] in which the angioparalytic attacks used to run a milder course than the sympathico-tonic, and especially with less vomiting. I have observed a case of this description, in which I was able to demonstrate in some attacks an increase of temperature in the auditory meatus of the affected side, in others a diminution of temperature.

In many cases, which present in other respects the sympathico-tonic or the neuro-paralytic type, the oculo-pupillar symptoms are entirely absent. Finally, there are cases of migraine which seem to present no local vaso-motor disturbances, in which, especially, no difference in the color and temperature of the two sides of the head can be shown to exist during the attack.

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[1] L. c. p. 335.

The duration of the attacks is, as a rule, from a few hours to half a day; they more rarely last a whole day or several days, with alternations of severity. The gradual diminution of the pain, which marks the termination of the attack, is apt to occur towards evening; the patient then usually feels exhausted, and falls into a sleep, from which, in the majority of cases, he awakes free from pain. The attacks usually occur at considerable intervals of time, and quite often repeat themselves in a very distinct type, seeming to have a preference for intervals of three or four weeks. In the female sex the paroxysms often, but by no means always, coincide with the appearance of the catamenia. The intervals, as a rule, are quite free from pain, or other morbid symptoms, except that there is sometimes a tenderness in the region of the ganglion supremum and of the spinous processes. In the atypic cases, and sometimes also in those which retain a strict type, bodily and mental exertions, mental disturbance (especially anger), as well as drafts of air, inequalities of temperature, the use of warm drinks, and disturbances of digestion, may aggravate or even bring on an attack.

Analysis of the Symptoms.

For reasons to be explained hereafter, it seems expedient not to begin the analysis with the cardinal symptom, pain, but with the *vaso-motor and oculo-pupillar symptoms* above sketched.

The group of symptoms called *hemicrania sympathico-tonica* is to be explained by supposing (as is implied in the name, given by du Bois-Reymond) a *unilateral tonic spasm of the vessels of the head, caused by tetanus in the cervical region of the sympathetic, or in the spinal centre of the cervical sympathetic.* The condition of the temporal artery, the anæmia of the face, the fall in temperature, the sunken eye, show that the blood-vessels of the suffering half of the head are in a state of tonic contraction at the height of an attack. If the cause which produces the contraction is taken away, then the excessive effort is followed by a state of relaxation, in which the vessels give way to lateral pressure more than usual. This secondary relaxation explains the redness of the conjunctival mucous membrane, the lachrymation, and the redness and heat of the ear which occurs towards the close of the attack. The tendency to vomit, which very often indeed goes with this form of migraine, can be explained by the variations in intracranial blood-pressure, which are a necessary accompaniment of the fitful contractions of the vascular muscles, alternating with partial relaxation, as is usual in tonic spasms.

A supposed tonic cramp of the vascular muscles, affecting the one side of the head in this manner, can only originate, according to our knowledge of physiology, in the sympathetic nerve of the corresponding side, or in the spinal centre for the fibres of the sympathetic—that is, in the corresponding half of the cilio-spinal region of the cord. This form of migraine is therefore to be referred to a *morbid state of the cervical sympathetic, or of the corresponding region of the cervical cord, which is liable to periodic aggravations.* In favor of this hypothesis the following reasons may be urged with special force:

1. *The alterations in the pupil.*—The dilatation during the height of the attack depends on an increase in the tonic excitation of the dilator fibres, which arise from the cilio-spinal centre and follow the course of the cervical sympathetic; the subsequent contraction depends on a secondary diminution in innervation, corresponding to the condition of the vaso-motor fibres.

2. *The local sensitiveness in the region of the ganglion cervicale supremum (sometimes also the g. c. medium), and at the spinous processes of the lowest cervical and uppermost dorsal vertebræ*, corresponding to the regio cilio-spinalis of the cord; occurring during the attack, and sometimes even in the intervals of freedom from pain.

3. It is also proper to mention the *increase in the salivary secretion* (which is sometimes greater, sometimes less; Berger saw over two pounds discharged in a single attack), and its *increased viscidity;* for there are secretory fibres destined for the salivary glands which follow the course of the cervical sym-

pathetic, experimental irritation of which produces a parallel effect in animals.

The objection made by Brown-Séquard and Althann against the supposition of a unilateral tetanus of the vessels of the head, namely, that Kussmaul and Tenner's experiments have proved that arterial anæmia of the brain must produce epileptic spasms, cannot be sustained; for when the cervical sympathetic is actually irritated in experiments, or its central end is tetanized, we see only the symptoms of unilateral contraction of the vessels, and lowering of temperature without convulsions. The same is the fact in pathological irritations of the sympathetic of unquestionable character in the human subject.[1] Probably in hemicrania sympathico-tonica there is nothing like an equal anæmia of the entire half of the brain, but rather a temporary inequality in the circulation of the several provinces and regions—perhaps especially in the case of the layers of the cortex, which are supplied from the vessels of the pia.

The symptoms of *hemicrania angio-* or *neuro-paralytica* are to be referred to a condition the reverse of the preceding—a condition of *relaxation of the blood-vessels in one half of the head, caused by a diminished innervation of the vessels, and therefore due to a lessened action on the part of the corresponding cervical sympathetic or its spinal centre.* The redness, heat, and swelling of the side of the face, the injection of the conjunctiva, the lachrymation, the redness and increased temperature of the ear, the increased secretion of sweat, the occasional ephidrosis unilateralis, the dilatation of the temporal artery and carotid, the dilatation, demonstrated in some cases, of the vessels of the fundus oculi, are easily explicable by the relaxation and increased fullness of the vessels of the head, by the arterial hyperæmia due to diminution of the activity of the vascular nerves. Whether this relaxation of the vessels of the head may be preceded by a stage of primary contraction, of spastic cramp, perhaps of very short duration, has as yet neither been proved nor disproved by any direct observation.

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[1] Cf. *Eulenburg* and *Guttmann*, Die Pathologie des Sympathicus. Berlin, 1873, page 3 et seq.

The hypothesis of affection of the cervical sympathetic or its spinal centre in hemicrania angio-paralytica is supported, not only by the local tenderness of the sympathetic and the spinous processes, which is often demonstrable, but more particularly by the concomitant *oculo-pupillar* symptoms. The contraction of the pupil depends on a loss of energy in the dilator fibres which run in the cervical sympathetic; the narrowing of the palpebral fissure, the retraction of the bulbus, the occasional ptosis, depend on a loss of energy in the smooth (non-striated) muscles of the eyelids, discovered by H. Mueller (especially the palpebralis superior and the musc. orbitalis).[1] These symptoms are well known as following the experimental section of the sympathetic in animals, and as accompanying a great variety of pathological conditions in man, which involve an interference with the conductivity of the cervical sympathetic (as inflammation, compression by tumors, wounds, especially those that divide the nerve, etc.). It is impossible to go farther into this point at present.

The *retardation of the pulse* during the attack is probably a symptom of partial hyperæmia of the brain due to relaxation of vessels, or of the consecutive anæmia of other regions of the brain, especially the medulla oblongata. According to the investigations of Landois, retardation of the pulse occurs both in artificial anæmia and in the (venous) hyperæmia of the brain produced by compression of the superior vena cava; this is the case even after extirpation of both cervical sympathetics, but not after destruction of the medulla oblongata or section of both vagi. This retardation of the pulse, which in case of a maximum of cerebral hyperæmia may go to the extent of arresting the heart and may be connected with epileptiform convulsions, depends, as Landois has shown, upon a direct, not a reflex irritation of the medulla oblongata and the vagi; section of the latter in the period of hyperæmic retardation of the pulse is directly followed by increased rapidity of the pulse.[2]

The medulla oblongata being also the centre of most of the vaso-motor nerves of the body, it follows that an irritation of this important part of the brain is entirely adequate to explain the small and contracted radial artery observed in this sort of cases (Moellendorff), the icy coldness of hands and feet, not to be relieved, the chilly sensations felt over the whole surface of the body, and, finally, the suppression of the perspiration during the attack, often with the sole exception of the affected side of the head. The contraction of the peripheral arteries caused by the increase of tone is followed, as always, by a stage of dilatation, of secondary relaxation. The latter condition may explain the phenomena of increased secretion of saliva and urine, as also the swelling of the liver and hypersecretion of bile described by Moellendorff, and the increasing plethora of the abdominal organs and inclination to broncho-tracheal catarrhs and emphysema of the lungs. (See below.)

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[1] Cf. *Horner*, Ueber eine Form von Ptosis. Monatsbl. f. Augenheilkunde. 1869. VII. p. 193. *Nicati*, La paralysie du nerf sympathique cervical. Étude clinique. Lausanne, 1873.

[2] Centralbl. f. d. med. Wiss. 1865. No. 44; 1867. No. 10.

If we now turn to the cardinal and pathognomonic symptom of hemicrania, the *unilateral pain, occurring only at intervals*, we encounter the double question: Where, in what regions of the peripheral or central nervous system, does the hemicranial pain originate? And how does it originate? Unfortunately, we cannot answer the first question at all, at the present time; the second we can answer only hypothetically.

As regards the seat of pain, we have already pointed out that most of the older, and many of the later authors place it in the cutaneous (frontal) branches of the first division of the trigeminus; yet, so great a number of points have been shown in which the neuralgias of these branches of the trigeminus differ from hemicrania, that the above view can hardly be regarded as admissible. If it be assumed that the trigeminus is concerned in the origination of the pain, we ought, I think, at all events to restrict our assumption to those branches which go to the dura mater, and which, by the way, arise from all three divisions of the trigeminus, namely: the nervus tentorii of Arnold, from the first division, which passes through the tentorium to the sinuses, a branch running with the arteria meningea media from the second division, and the nervus spinosus of Luschka from the third. The possibility that these nerve-branches may be implicated in hemicrania can neither be directly disproved nor directly proved; at the most, the character and apparent localization of the sensation of pain (see above) might be regarded as supporting the view that it originates within the dura mater. We know nothing certain of the nerves of the arachnoid; but in the pia numerous nerves are found, following the vessels in the form of plexuses, and some of them entering the cortex along with the vessels (Koelliker). These nerves originate in part from the vertebral and carotid plexuses of the sympathetic, but partly also from cerebral nerves at their exit (Bochdalek), and especially from the trigeminus. Probably all or most of these nerves must be regarded as vascular nerves; and probably we are not in error in ascribing to them, as we shall immediately explain, a considerable part, whether direct or indirect, in the origin of hemicranial pain.

Romberg's view, which located the pain in the cerebral mass, had no other essential foundation than the "associated sensations" occurring in the region of the fifth nerve and the nerves of sense, and the manifest injurious influence of moral and mental exertions. The uncertainty of these foundations is evident; Hasse[1] also remarks, and correctly, that, judging from the analogy of the other neuralgias, "associated sensations" of several cerebral nerves and reflex phenomena caused by them are no reason for refusing to regard the intracranial and meningeal branches of the trigeminus as equally the seat of pain.

In cases of hemicrania sympathico-tonica, du Bois-Reymond first proposed the theory that the *tonic spasm of the smooth muscular fibres of the vessels themselves* was what was felt as *pain*, after the analogy of those painful sensations which occur in the striped muscles in spasm of the calves, or in tetanus, and in the smooth muscles of the uterus in labor, the intestine during colic, etc., or as the skin is found to be painful in the cold stage of intermittent fever. Probably this pain is caused by pressure upon the nerves of sensation that are distributed in the muscles; this pressure, and in consequence the pain also, will increase when the tetanized muscles are subjected to an increase of tension, as is, for instance, the case in spasm of the calves when the muscles are stretched by means of their antagonists, or by supporting the soles of the feet and resting the weight of the body on the latter. The same effect will be produced, in case of tetanus of the muscles of the vessels, by an increase of lateral blood-pressure within the vessels. *Thus the observation that the pain increases with the rise in blood-pressure, and simultaneously with the pulsations of the temporal artery, finds a rational explanation.*

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[1] Krankheiten des Nervensystems. 2d Ed. p. 73.

This ingenious theory of du Bois-Reymond's does not fit cases of hemicrania neuro-paralytica, nor those cases of migraine which are unaccompanied by any marked vaso-motor disturbances. It seems to me, therefore, that another interpretation of the pain is preferable—one applicable to the latter cases as well as the former, and less indirectly. The variations in the arterial supply, the temporary anæmia or hyperæmia of the half of the head, might furnish a cause for irritation of the sensitive nerves of the head, either in the skin, the pericranium, the meninges, the sensitive regions of the brain itself, or in all these parts together, and such irritation might cause the hemicranial paroxysm. That nerves of sensation are intensely excited, and react with pain when the calibre of the vessels that accompany and irrigate them is altered, and especially when this alteration takes place with some suddenness, is a fact not rarely observed in a great variety of neuralgias (prosopalgia, sciatica, etc.); the neuralgias which zoster causes—principally in the trunk, but also in the face and extremities—may be referred with great probability to this source; and, in general, local and systemic anomalies in circulation may be looked on as one of the most important causes of neuralgic affections in the nerves of almost any part. The increase in the pain of hemicrania upon bowing forward, coughing, etc., the peculiar influence of compression of the carotids, are likewise explained by the variations of intracranial blood-pressure. The case above described, in which the pain increased when the carotid *of the same side* was compressed, and diminished when that of the *other* side was compressed, shows very strikingly, at any rate, the favoring influence of local anæmia. *Probably, in migraine, the local anomalies of circulation, without regard to their special mode of origin, are to be regarded* *as the essential and universal causal condition, while, on the other hand, tetanus or relaxation of the muscles of the vessels exercises rather an indirect influence, confined to single cases, and acting through the local anæmia or hyperæmia of which it is an important cause.* The inequality and inconstancy of the oculo-pupillar and of the vaso-motor phenomena as well, speak loudly in favor of this view. It cannot appear at all unaccountable that, in the angio-paralytic and hyperæmic form of migraine, temporary increase of blood-pressure, increased fullness of the small arterial and venous vessels, should act as an irritant upon the sensitive nerves exactly as the opposite condition of vascular spasm and local anæmia does. Experiments have proved that diminution and increase of the supply of blood, local hyperæmia and anæmia, agree in many other respects in their mode of action ; that, for instance, the well-known epileptoid attacks occur not only in anæmia of the brain (as in the experiments of Kussmaul and Tenner), but also in hyperæmia of the same organ, caused by arrest of the venous current discharged from the brain by closure of the vena cava superior ;[1] and that, in like manner, the effect upon the heart's activity and the rate of the pulse is quite analogous in both cases.

If the interpretation of the hemicranial pain here given is the correct one, it explains also some of the minor symptoms, such as the *paralgiæ in the province of the nerves of sense*, which may with probability be referred to irritation of their respective central organs (or perhaps of their peripheral apparatus?) in consequence of the periodic vacillation in the supply of blood. The *cutaneous hyperalgesia*, the *hyperpselaphesia* observed in a few cases (Berger), are likewise referable to the change in the amount of blood held by the skin, especially in the case of arterial hyperæmia of the latter. The *tendency to vomit* was referred by du Bois-Reymond to fluctuations in the blood-pressure within the brain, which, however, may be due, not solely to spasm of the vessels, increasing and relaxing by turns, but also to some other causes of a mechanical nature capable of producing anæmia or hyperæmia. The *ecchymoses* in the conjunctiva of the corresponding eye, observed by Berger as accompanying hyperemesis, depend probably upon the mechanical action of violent vomiting, during a period of changed tension in the vascular walls that predisposes them to rupture. The *watery stools* observed at the close of certain attacks are probably due, like other excesses of secretion that occur at this time, to the general condition of secondary exhaustion of the vaso-motor nerves after spasm of the latter. In this synopsis many special points have to remain comparatively neglected for want of space.

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[1] *Hermann* and *Escher* in Pflu·ger's Archiv. 1870. p. 3.

[2] *Landois*, l. c.

Course and Prognosis.

The course of migraine is very chronic, with hardly an exception. The disease may last a lifetime, or the greater part of one, sometimes with scarcely any change in severity, sometimes with an increase or diminution. But it very often happens that, with the advance of age—say after the fifty-fifth year of life—the attacks gradually become less frequent, and even wholly cease; the climacteric years of women seem especially to exercise a favorable influence in this respect. In a few cases the malady disappears at an earlier period spontaneously, or under the influence of remedies. This I have observed chiefly in young persons who were free from any demonstrable predisposition, such as hereditary tendency, etc.

The *prognosis* of hemicrania may be called favorable, in so far as it never directly produces any severe effects which seriously threaten health or life. Although Moellendorff says that a "plethora of the abdominal organs" and a great tendency to broncho-tracheal catarrhs and emphysema of the lungs are developed in all persons who are subject to migraine, yet it must be said that this is a most exaggerated statement; the matters it refers to are not properly the consequence of migraine as such, but are co-ordinate effects of the same cause—namely, of those local and general disturbances of circulation which we have learned to recognize as prominent factors of hemicrania.

On the other hand, the prognosis is decidedly unfavorable as regards the disease itself. A spontaneous disappearance is rarely to be hoped for in youth, and not with certainty in old age. The results of treatment are, upon the whole, very unsatisfactory, yet it must be admitted that of late our improved knowledge of the causes of the complaint has enabled us to enter upon a rational plan of treatment, which is somewhat more successful. Old cases, and those dependent on a hereditary taint (neuropathic predisposition), present of course the least hope of a cure, either spontaneously or by treatment.

Treatment.

A treatment directed to the *causes* of hemicrania, in our present state of ignorance of these causes, is but too plainly impossible. As for those special cases in which a unilateral tetanus or paralysis of the vessels of the head produces the attacks of pain, the causes which periodically excite or depress the cervical sympathetic or its spinal centre, and the nature of the sympathetic affection itself, are entirely obscure at present.

The treatment required by the disease is partly general—that is, corresponding at least in theory to the indicatio morbi ; and partly symptomatic or palliative, consisting in attempts to conquer the single attacks. Of the general treatment, the greater part depends on empiricism ; of the palliative, a few points, deserving of attention at least, are rationally derived from late pathological discoveries. It is, however, not always easy to draw a sharp line between these two classes of treatment, in the case of the remedies most in use.

Among the great number of remedies administered empirically, the preparations of iron, quinia, and caffein are by far the most popular, and certainly not without reason, although the universal agreement in praising them seems to show that they have usually been adopted without clear views, or even with quite wrong views of their action. The preparations of *iron*, especially the carbonate, so much praised by Hutchinson, Stokes, and others, are hardly specifics against migraine, but may serve to improve the constitution of anæmic and weakly persons who are victims of migraine as of other forms of neuralgia. In recommending *quinia*, as also such analogous remedies as *quinoidin* and *bebeerin*, the anti-periodic effects of the remedy have been chiefly regarded, and the tolerably regular recurrence of the attack has encouraged a hope of corresponding success with the remedy. But experience shows that the use of quinia, no matter in what form, does not ordinarily affect the regular periodicity of the attacks, especially when they occur at wide intervals; but that a considerable dose of quinia (from seven and a half to eighteen grains), given once or more often, may sometimes shorten an attack or arrest it at once. It is possible, as A. Bernatrik[1] and others have pointed out, that this favorable effect of quinia, and the analogous action of caffein, depend chiefly on the fact that the drugs produce an increased activity of the vaso-motor nerves, an elevation of arterial tension, in cases where this is pathologically depressed. At least, according to some observations, the quinia seems to have the greatest effect in the angio-paralytic or neuro-paralytic form of migraine.[2]

Caffein is given either pure or in the form of citrate, which is really only a mechanical mixture of caffein and citric acid; and usually in pastilles, containing from one-half to one grain each. This is probably the most popular prescription in migraine. I must confess that the continued use of caffein in the inter-paroxysmal periods seems to me, like that of quinia, to be of less value than the single or repeated large dose of one or two grains before or during the attack. The same is the case with the subcutaneous administration of caffein, which I have tried repeatedly. The *guarana paste*, which has been much praised as a specific, and recently by Wilks[3] among others, and which has been needlessly incorporated into the German pharmacopœia, is known to contain as its active principle guaranin, which is perfectly identical with caffein. Thanks to the puffs it has received, it still retains a special popularity with those who suffer from migraine.

These facts entitle us to place a special confidence in the action of another remedy, in cases of the angio-paralytic form. This remedy is *ergotin* (aqueous extract of ergot), which we know to produce contractions of the blood-vessels; an action which, according to Wernich, Holmes, P. Vogt,[1] and others, is probably effected by means of the vaso-motor centre in the medulla oblongata. I have lately used this remedy (which Woakes[2] recommends very highly), both in migraine and in the non-unilateral cephalalgia vasomotoria,[3] with decided success, in doses of from nine to fourteen grains daily in the form of pills. Berger[4] employed the remedy in the form of subcutaneous injections in two cases of angio-paralytic hemicrania, with a repeatedly favorable action upon the symptoms.

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[1] Wiener Med. Presse. 1867. No. 28.

[2] Cf. *Eulenburg* and *Guttmann*, Pathologie des Sympathicus. p. 26.

[3] British Med. Journ. April 20, 1872.

I will mention only a few other remedies — some of recent renown, others of an old fame newly revived. Such are strychnia, arsenic, nitrate of silver (Clifford Allbutt), sulphate of nickel (Simpson), bromide of potassium (Ferrand, J. D. Davis), chloride of ammonium (Anstie), oil of turpentine (Warburton Begbie), lupulin (Huguier). In former times the "digestives" were popular, especially the great multitude of bitter and aromatic remedies. The causal relations of migraine to disturbances of digestion, especially to those of the stomach, which were said to be removed by the latter remedies, are quite as problematical as are the curative successes of the drugs in the presence of the actual disease.

Among the *spas* and *water-cures*, the best reputation is possessed by the *iron springs* and *iron-moor-baths* (Pyrmont, Franzensbad, Schwalbach, Reinerz), and the *sea-bath;* the reputation is not undeserved, though the benefit is usually for the most part transitory. The continued use of the treatment in *cold water establishments*, and the residence in lofty *mountain regions* (as St. Moritz, where there are also iron-springs), have often proved of benefit in my experience.

In treating the single attack, it has long been known that certain *precautionary measures* are indispensable, in order to exclude as far as possible external sources of irritation, and to ensure mental and physical quiet. The posture should be that of rest, and in the anæmic form the patient should lie flat on the back, with the head a little raised; she should remain in a moderately lighted chamber, noises and disturbance of every sort being kept away. These are indispensable precautions during an attack of hemicrania, and they usually diminish its severity and duration. The administration of *palliative medicines*, however, is found less useful upon the whole than in other forms of neuralgia; sometimes they seem even to do harm, by annoying and disquieting the patient, who often longs for nothing more than to be let alone, knowing full well from her own experience and that of others how uncertain is the effect of remedies. In such cases, therefore, we should avoid the useless and unwelcome πολυπραγμοσύνη, which is so unsuitable to the character of a scientific physician. Among the older palliatives, *cold* and *compression* often do some good, though but to a very slight and temporary extent. It is very proper to keep an ice-bag applied for a long time to the forehead and temple. The weight of the bag has also an advantage in the compression it exercises; for this reason, and because of its superior cooling effect, it finds no substitute in cold applications or even ice-cloths; besides, all wet applications have to be changed often, wet the patient, and therefore, if they do not produce a very speedy and convincing effect, must be soon given up.

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[1] Berliner klin. Wochenschrift. 1872. No. 10.

[2] British Med. Journ. 1868. II. p. 360.

[3] Berliner klin. Wochenschrift. 1873. No. 15.

[4] L. c. p. 330.

Compression of the head against a firm body, by the supporting hand or by a cloth wound firmly about the head, are palliative remedies, known to most patients, but very slight and transient in their effects. Of much more certain result, in the cases before described, is the *compression of the carotid*, a procedure which only the physician himself can execute, which most patients bear unwillingly, and but a short time, and which relieves only for so long as it is actually applied.

In regard to the use of *narcotics* during the attack, the opinion expressed concerning palliatives in general will apply—whether the administration by the mouth or the subcutaneous syringe is preferred, whether opium and its alkaloids, or belladonna and similar remedies are tried. The comparatively small benefit of hypodermic injections in hemicrania, as compared with their success in other neuralgias, must be ascribed partly to the circumstance that in this case no single nerve-branch or cutaneous nerve-district is affected, so that the favorable local action of the narcotic is of no advantage. Thus, *ex juvantibus et non juvantibus*, it is often easy to distinguish a hemicrania from a frontal neuralgia. If a few physicians have observed very good results, and sometimes a permanent cure, from the injection of morphine in hemicrania, it is possible that in such cases there may have occurred a confounding of hemicrania with symptomatic headache of another sort, or with frontal and temporal neuralgias.

The epidermic application of narcotics and anæsthetics (*e. g.*, friction with ointment of belladonna or veratria, rubbing the head with pomade of chloroform, according to Cazenave's directions) is certainly of still slighter value, though not wholly doubtful.

Of the symptomatic action of quinia, caffein, and ergotin in the angio-paralytic form of migraine, we have spoken before. On the other hand, another and a very recent remedy seems to play an important part in the sympathico-tonic form, namely, the *nitrite of amyl*. The indication for its use depends on the fact that it possesses the power of dilating the blood-vessels, although whether by acting on their contractile elements (Richardson,[1] Lauder Brunton,[2] Wood[3]) or by paralyzing the vaso-motor system (Bernheim[4] and others), is as yet unsettled; when inspired, it causes almost instantly an intense reddening of the face, a feeling of great heat in the head and face, injection of the conjunctiva, great acceleration of the pulse, with diminished tension of the radialis; if the inhalation is continued, symptoms of fainting may easily supervene. Berger[5] first used the nitrite of amyl in a case of migraine, evidently of the sympathico-tonic form, with almost instant effect; the pain was "charmed away," as it were, and did not return during the day. Vogel and Holst,[6] and I myself also, have seen the momentary disappearance of the pain in cases that presented the features of vascular spasm, but the pain returned in most instances after some time. In these inhalations the greatest care must be taken, especially if the patient is anæmic; beginning with a dose of one drop, we may by degrees rise to three or five drops, and in case of need repeat the inhalation after a time. Anything more than a palliation of the trouble I have never seen; but Holst states that in a female patient the attack itself was not only cut short, but the following attack was postponed longer than usual.

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[1] Med. Times and Gazette. 1870. II. p. 469.

[2] Arbeiten des physiologischen Instituts zu Leipzig. 1869. p. 101.

[3] American Jour. of Med. Science. July, 1871, p. 39, and Oct. p. 359.

[4] Pflueger's Archiv f. Phys. VIII. p. 254. Compare also *Eulenburg* and *Guttmann*, Zur Kenntniss und Wirkung des Amylnitrits. *Reichert's* und *du Bois-Reymond's* Archiv. 1873. p. 441. *Pick*, Centralblatt. 1873. No. 55.

[5] Berliner klin. Wochenschr. 1871. No. 2.

[6] Dorpater med. Zeitschrift, 1871. II. p. 261.

Holst stated, as the result of observation in his own case, that in pronounced attacks of migraine the free use of any warm drink gave relief at the moment when general perspiration broke out. It is necessary to remind the reader that in many cases of migraine the use of warm drinks provokes or aggravates the attacks. The inhalation of carbonic oxide gas, praised by A. Mayer,[1] may perhaps derive its good effects from the known fact that it paralyzes the vaso-motor nerves, and thus may probably remove a temporary spasmodic condition of these nerves.

Another modern remedy of great importance, both in relieving the symptoms of various forms of migraine, and perhaps also in accomplishing a permanent cure, is the *constant galvanic current.*

This remedy seems to have a destiny, such as hardly any other possesses, in the treatment of hemicrania, for it places in our hands the power of exercising a real and powerful influence, locally limited, regulable in respect to quantity and quality, upon the cervical sympathetic and the upper regions of the spinal cord in the human subject. The electrical method, based in part upon du Bois-Reymond's and Moellendorff's theories, and in part developed independently, has therefore accomplished much in conquering the disease, and has successfully adopted certain empirical procedures in its treatment. Examples are furnished by Benedikt,[2] Frommhold,[3] Fieber,[4] M.

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[1] Wiener med. Presse. 1865. No. 46.

[2] Elektrotherapie. Vienna. 1868 [and new ed. in 1875].

[3] Die Migraine und ihre Heilung durch Elektricität. Pesth, 1868.

[4] Compendium der Elektrotherapie. Vienna, 1869.

Rosenthal,[1] Althaus,[2] and others. Holst[3] was the first to carry out practically, in connection with the polar method of Brenner,[4] a really methodical and rational application of the constant current, based on the *diagnosis of the several forms of migraine*, a point upon which I insisted as necessary, several years ago.

Holst's method consisted in placing one electrode, the one from which special action was expected, upon the cervical part of the sympathetic, at the inner edge of the sterno-cleido-mastoid muscle—this electrode is long and narrow and has considerable surface; the other one is placed on the palm of the hand, and the circuit closed. In hemicrania sympathico-tonica the pole on the neck was made positive; the current (from ten to fifteen elements) was closed suddenly, and after a passage of two or three minutes was gradually stopped. In hemicrania neuroparalytica, on the contrary, the negative pole was applied to the above spot, while the current was not only suddenly closed in the metallic part of the circuit, but was made to produce a powerful excitation by means of repeated closures and openings, or in some cases by reversals. The former procedure, intended to bring about a direct diminution of the excitement, was used more commonly by Holst, especially in cases where the condition of the muscles of the vessels was uncertain; for he regards as the primary cause of every hemicrania, even if it manifests itself in its secondary stage of paralysis, an abnormal excess of excitability of the vaso-motor nervous system of certain vascular territories (or cerebral regions); and when this abnormal excitability is lessened, it is probable that the disposition to a secondary stage of relaxation of the vascular walls is removed. The observations made by Holst, some thirty in number, are very much in favor of the practice above described. Usually a sense of comfort and relief came in a very short time; in a few cases there was a lengthening of the intervals between attacks.

The *induced current* has also been recommended, especially by Frommhold and Fieber. The former prefers the primary induced current, and applies one of the poles to the median line of the back of the neck, high up, the other upon the forehead or the arcus superciliaris. Fieber recommends the use of the so-called electric hand: the patient takes one conductor in his hand, the operator holds the other in his left hand, while pressing the palm of his right firmly upon the patient's forehead, which is previously wetted. Fieber says that this treatment seldom fails, and sometimes produces surprising results. Althaus, on the contrary, found faradization useless in most cases, while the passing of the constant current continuously through the head was of value.

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[1] Handbuch der Diagnostik und Therapie der Nervenkrankheiten. Erlangen, 1870.

[2] Treatise on medical electricity, etc. 3d edition. London, 1873.

[3] L. c., p. 275 et seq.

[4] Lehrbuch der functionellen Nervenkrankheiten. p. 131.

ANGINA PECTORIS.

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WE designate by the name of angina pectoris a group of symptoms, of which the most characteristic are the following: *Pain in the region of the heart, occurring in paroxysms, which usually radiates over the left side of the thorax and the left arm, more rarely over both sides and arms; the pain is associated with a peculiar sensation of anxiety and constriction, and often also with other motor, vaso-motor, and sensitive disturbances. Stenocardia is another name for these attacks.* The period between the paroxysms is free from pain, and usually from all other symptoms. The disease, therefore, bears the plainest marks of a *neurosis*, and may be classed, according to its symptomatology, with the *visceral neuralgias*, including cardialgia, colic, hysteralgia, and so forth.

History.

Heberden gave it the name of angina pectoris, from the torturing anxiety (angina from angi; badly translated by Herzbraeune, in German). At the same time, or a little earlier (1768), the disease was described by Rougnon. Later observers described it under the following names, now antiquated: Asthma convulsivum (Elsner, 1778), Asthma dolorificum (Darwin, 1781), Diaphragmatic Gout (Butler, 1791), Asthma arthriticum (Schidh, 1793), Syncope anginosa (Parry, 1799), Sternalgia (Baumes, 1806), Sternocardia (Brera, 1810), Pneumogastralgia (Téallier, 1826), Cardiodynia (Baumgaertner, Harless). The majority of these synonyms show ignorance regarding the true nature of the disease; they lay weight sometimes on the apparent seat of pain, sometimes on the analogy with attacks of asthma or syncope, sometimes on the supposed connection with arthritis, which was especially noted by English observers. Parry first called attention to the coincidence of stenocardiac attacks with ossification of the coronary arteries of the heart, and Percival in 1773 to the coincidence with diseases of the abdominal organs. Testa, Brera, Latham, and others explained the attacks as due to enlargement or dislocation of single organs of the abdomen, which pressed mechanically against the heart.

Most later pathologists have either assumed an exclusively nervous origin for the disease, or else have assumed the existence of a nervous, dynamic form in addition to the organic, or the one complicated with heart-disease; of the former, the origin was assigned to a great variety of nerve-trunks more or less remotely connected with the heart. Sometimes the phrenic nerve (Bouillaud), sometimes the intercostals (Jolly), were thought chiefly implicated; but the vagus (Lartigue, Desportes, Chapman) and the sympathetic (Lobstein, Laënnec) were the favorites, yet they failed to bring the phenomena of angina pectoris into agreement with the known functions of these nerves, which have been only very recently explained. The disease was regarded as essentially a *neuralgia of the nerves of the heart;* thus Laënnec called it neuralgia cordis, Trousseau an "epileptiform neuralgia," and Romberg and Friedreich a hyperæsthesia of the cardiac plexus. Others laid stress not only on the neuralgic character of the disease, but also on the accompanying symptoms, especially the paroxysmal alteration of the action of the heart; and thus Dommes and Jahn designated the condition as *paresis or paralysis*, Stokes[1] as a temporary increase of weakness, in a heart already weakened, with fatty change in its muscle (and with hyperæsthesia); Bamberger, contrary to Stokes, as increased action of the heart, *hyperkinesis*, with hyperæsthesia; von Dusch as hyperæsthesia, with spasms of the heart; Eichwald as over-exertion of the heart, due to mechanical obstacles to its activity; others (as Barkow) even called it a trophoneurosis of the heart, *cardioneurosis trophica.* Some autopsies of Lancereaux's (1866) pointed to an affection of the cardiac plexus; the aorta and the coronary arteries were, at all events, found altered in the same cases. Almost at the same time Cahen (1863) expressed the belief that angina pectoris must be classed with the *vaso-motor neuroses.* Although this assumption of Cahen's was quite untenable and incorrect, yet in some respects it gave a decisive turn to the controversy; he deduced the symptoms of angina pectoris from a vaso-motor neurosis accompanying intercostal or bronchial neuralgia—that is, from a spasm of the blood-vessels of the heart or the lungs. He was quite aware that there was absolutely no proof of this supposition at that time. Traube[1] thought it possible to refer certain symptoms of the stenocardiac attack (the rapidity of pulse and the diminished size with increased tension of the blood-vessels) to an increased excitement of the vaso-motor nervous centre. Landois (1866) first subjected all the symptoms of angina pectoris to a thorough and systematic physiological analysis, and divided the disease, in accordance with the results of this analysis, into four classes, caused respectively by: 1. Disturbance of the activity of the excito-motor cardiac nervous system; 2. Conditions of irritation in the region of the cardiac branches of the vagus; 3. Reflex excitement, due to irritation of the organs of the abdomen (*angina pectoris reflectoria*); 4. An affection of the vaso-motor nerves of all, or of most regions (*angina pectoris vasomotoria*).

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[1] Diseases of the Heart and Aorta. Dublin, 1854, p. 486.

Subsequently, in 1867, Nothnagel described some cases of angina pectoris vasomotoria, and Landois and I described it in our monograph upon the vaso-motor neuroses; finally, Guttmann and I examined into the part taken by the several nerves belonging to the cardiac plexus, and especially by the sympathetic.[2] The view of the nature and pathogenesis of the disease thus obtained, though very defective, was considerably enlarged by a series of physiological experiments upon the sphere of action of the cardiac and vaso-motor nerves (v. Bezold, Goltz, Bernstein, Ludwig, M. and E. Cyon), or, to speak more correctly, it was first rendered tenable by these experiments. Pathological anatomy has as yet contributed little that is of use;

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[1] Symptome der Krankheiten des Respirations- und Circulationsapparates. S. 41.

[2] Pathologie des Sympathicus, in Griesinger's Archiv f. Psychiatrie. p. 688.

angina pectoris belongs to that class of neuroses which show most convincingly how much we owe to the discoveries of experimental physiology and their pathological application, and how far mistaken in point of fact those are who expect to find in pathological anatomy the source of every improvement in neuropathology. Perhaps no ray of light would ever have penetrated the obscurity in which this disease was wrapped if Physiology had not shot forth those lightning-flashes of hers, which threw such a wonderful illumination upon the whole matter.

Etiology.

Only a few scanty indications of the etiology of angina pectoris can be given. We do not here, nor subsequently, include in this statement the stenocardiac attacks which are properly symptoms of certain chronic diseases of the heart, such as insufficiency of the aortic valves, stenosis of the aortic orifice, fatty degeneration of the heart; nor those other cases due to the circumstance, hardly possible to distinguish during life, of ossification of the coronary arteries. The etiology of these cases is that of the cardiac diseases named, and of endarteriitis deformans. But if we confine ourselves to the uncomplicated cases—those of angina pectoris in the narrower sense, and especially angina pectoris vasomotoria, we have but very few points upon which to base our etiological views. It has been considered (after the formerly prevalent fashion) that the dyscrasiæ constituted predisposing influences; especially was this thought to be the case with arthritis, and even a so-called hemorrhoidal diathesis. As for the supposed connection with arthritis, the case is probably one of coincidence of arthritis with endocarditis and endarteriitis, and the stenocardiac attacks referable to organic diseases of the heart or ossification of the coronary arteries. A connection between arthritis and the pure angina pectoris cannot be established upon any theoretical ground, and the cases contained in recent literature, which have been more closely sifted, do not show a trace of such a relation. It were superfluous to lose a word in speaking of the hemorrhoidal diathesis. It seems more reasonable to ascribe a favoring influence to anæmia; at any rate, we see a greater frequency of angina pectoris, as of many other neuralgias (*e. g.*, intercostal), in anæmic persons. In other cases a hereditary tendency is unmistakable; this fact and many other circumstances connect the disease with the group of constitutional neuropathies, not to mention the peculiar connection often existing with other diseases of the group, such as hysteria, insanity, and epilepsy. Attacks of angina pectoris may form a symptom of hysteria; they may herald the epileptic attack, or may alternate with it; they may precede the outbreak of maniacal or melancholic conditions, or may constitute an intercurrent symptom of well-marked and long-standing disease of the mind. The nature of the etiological connection here existing is certainly quite hidden from us; the terms we are compelled to put up with for the present, as neuropathic constitution, congenital preformation, etc., give us no explanation, but only formulate our ignorance with precision; at the best, they are but hints for future investigation.

The possible predisposing influences of age and sex cannot yet be established with sufficient certainty. It is unquestionably true that angina pectoris, without hereditary tendencies or constitutional anomalies, may occur at an advanced age. But when it is said that the disease belongs chiefly to advanced age, the statement seems to rest upon a confounding of the true angina with stenocardiac attacks due to cardiac anomalies, atheromatosis, etc. The male sex is attacked far oftener than the female—Forbes says, in the proportion of 11 to 1—perhaps because it is more exposed to the influences which we are about to mention.

As a direct cause of the disease, even in persons predisposed, we must regard the influence of *exposure to cold*. Some good observations are decidedly in favor of this view; thus, for instance, Huebner saw a case in which the attacks appeared for the first time in a robust man of seventy-four, after a long ride in a post-wagon in cold weather. The harmful influence of damp and cold residences is especially mentioned by Nothnagel. Besides, the *excessive smoking of tobacco* seems to deserve to be regarded as an etiological factor. Beau describes eight cases in which the attacks ceased when smoking was stopped, and returned when the patients began to smoke again. The like is reported by Savalle, Championnère, and Blatin, and I myself have made out several quite analogous cases; I have also observed very exquisite cases of angina pectoris in a young anæmic cigar-maker, who had smoked a large number of strong cigars daily for several years. Finally, certain morbid conditions of the thoracic organs, by involving the nerves of the cardiac plexus, and morbid conditions of the abdominal organs, probably by irritating, in a reflex manner, the sensitive nerves of the abdomen, may produce the symptoms of angina pectoris. (See below, "Anatomical Alterations," and "Analysis of Symptoms.")

General Description and Course of the Disease.

Angina pectoris must be described as composed of attacks, separated from one another by longer or shorter intervals. The intervals are usually free from symptoms. They usually begin rather suddenly, without any other premonitory signs than pain, which begins at the region of the scrobiculus cordis or the lower part of the breast-bone, and shoots out sometimes over the left side of the chest and neck, and sometimes up along the sternum to the left arm or to both arms. In the latter case the pain is less in the right side of the chest and the right arm than it is in the corresponding part of the left side. With the pain, which is often called "constrictive," there is associated a peculiar feeling of anxiety, of impending death—a feeling which is not rarely met with in other visceral neuralgias, such as cardialgia and colic. The suddenness with which the attacks come on is especially striking; the patients are not rarely overtaken by it while walking or at work. The duration of the single paroxysm is usually short, commonly only a few minutes; but sometimes the attack does not end with the first paroxysm, intermissions or remissions being followed by repeated exacerbations, so that the entire attack is made up of a series of partial attacks, as is the case in many neuralgias.

Along with the disturbances of sensibility, disturbances of the action of the heart and of the movements of circulation and respiration are usually seen, which may present great variety. The beats of the heart are sometimes small, wanting in energy, intermittent, and even interrupted by considerable pauses; sometimes, again, they are increased, become violent, and are accompanied by a very great increase in the force of the beat. At the height of the attack of pain, the action of the heart seems usually to be weakened, or even arrested; in the intermissions or remissions, on the contrary, it is often much strengthened, or even extraordinarily violent. The pulse shows similar variations; in the radial artery it is sometimes small and of slight tension, sometimes strong and full; sometimes, even in the cases where the action of the heart seems increased, the radial arteries exhibit but a slight elevation and low tension. We often find at the beginning of the attack a cord-like hardness and contraction of the peripheral arteries, while later they are full and soft. Sphygmographic tests also show an increase in arterial tension in the beginning of the attack, but later a diminution (Lauder Brunton). The respiration also is hastened, is even violent, dyspnoïc, but sometimes retarded and superficial, or even quite arrested. But the latter symptoms are essentially effects of the pain. The patient dreads to take a full breath; if persuaded to try, he usually succeeds perfectly well. This dependence of the action of respiration upon pain has already been noticed by Parry. The respiration may also be quite free during the attack, as I have myself observed in some instances. The disturbances of the action of the heart and the circulation are shown by changes in the amount of blood present in the peripheral parts and in their temperature. During the attack the skin is often pale, cold, dry, almost bloodless in the hands and feet, the face pale and sunken; a general sensation of chill, chattering of the teeth, paralgic sensations (formication and prickling) in the tips of the fingers, may be caused by the cutaneous anæmia. Towards the close of the attack, or after it, there may be the contrary condition of redness, swelling, increased sense of warmth in the skin, and abundant perspiration may be secreted.

The course of the disease is almost always extremely chronic. The attacks recur (as in the case of epilepsy) at extremely variable intervals; they may sometimes be suspended for years; in some cases they may occur for a time every day without visible cause. I have never observed a regular periodicity in marked angina pectoris. As occasional causes, exposure to cold, bodily and mental exertion (especially exhausting bodily movement), and mental excitement, may be named as provocative; the attacks may sometimes be excited or made worse by pressure upon some points that are sensitive during the attacks, or constantly sensitive. Such points are the spinous and transverse processes of the cervical and upper dorsal vertebræ, and the region of the inferior angle of the scapula. It is rare that a diminution and spontaneous disappearance of the attacks occurs as time passes on; on the contrary, it is much more often the case that they grow worse and more frequent, the remissions being shorter and marked by exhaustion.

Anatomical Alterations.

We do not here describe those forms of angina pectoris which are complicated by faults in the valves of the heart, or fatty degeneration of this organ, nor those forms which are caused by atheromatous processes in the aorta, and especially by *ossification and contraction of the coronary arteries.* Anatomical conditions of the latter sort, usually connected with defects of the aorta or other abnormalities, have been described in earlier times by Hofmann, Jenner, Black, Parry, Birch, Kreysig, Ritter, Sluis, Ring, Crisp; in later times by Philipp, Wilks, Waldeck, Oppolzer, Fincham, Mason, Moorhead, Dickinson, Colin, Lockhart, Clarke, Haddon, Ogle, Grodsensky (Traube), and others. That these changes do not furnish the exclusive basis of the symptoms has been shown by the discoveries of Senac, Corvisart, Bianchi, and others, who even found extensive ossification of the coronary arteries without symptoms of angina pectoris having been observed during life; also by the negative results of examinations by Jucine, Johnston, Lentin, Erdmann, Desportes, Heusinger, Jahn, who have demonstrated a complete integrity of the coronary vessels in decided angina pectoris.

The nervous apparatus of the heart formerly received little or no attention at autopsies. The first positive statement seems to have been made by Heine (1841), who speaks of pathological changes in the cardiac branches of the vagus, and also in the phrenic nerve. The case was that of a patient treated by Skoda at the Vienna clinic, suffering with peculiar attacks, in the course of which the heart stood quite still for some seconds, usually during the period of from four to six pulsations, while the patient suffered from the feeling of inexpressible anxiety. At the autopsy, which was made by Rokitansky, the right phrenic nerve was seen involved in a dark blue hard knot, dotted with concretions of lime. Among the lax, pale gray cords that made up the plexus of cardiac nerves was seen the *cardiacus magnus*, which, rising from the plexus between the pulmonary artery and the descending aorta, just below the arch, entered into a black nodule of the size of a hazel-nut, and was thickened before entering the latter. The descending *branches of the left vagus* upon the anterior aspect of the left bronchus appeared similarly interfered with by a nodular blackish blue lymphatic gland that lay beneath them.

In connection with this an observation of Haddon's[1] should be mentioned, concerning the case of a man affected with angina pectoris, in whom the left phrenic nerve was found compressed by a bronchial gland of the size of a hazel-nut, situated close to the root of the left lung, and infiltrated with black pigment. The nerve-tubes at the spot seemed not interrupted, but slightly granular. The right phrenic nerve was normal, the vagi and recurrentes were also normal. The aorta was in a state of atheromatous degeneration and aneurysmal dilatation.

Anatomical changes in the cardiac plexus have been found by Lancereaux in a patient aged forty-five, who exhibited the usual symptoms of angina pectoris and died in one of the attacks. At the autopsy there appeared a contraction of the coronary arteries, and an alteration of the aorta at the point where the cardiac plexus lies upon it; the plexus was in a vascular condition. A few of the bundles of the latter were enveloped in exudation, and the outer sheath was thickened. Microscopic examination showed an abundant accumulation of round nuclei, which had pressed asunder and compressed the nerve-tubes; the medullary contents of the latter appeared grayish and granular. In two other cases Lancereaux found a change of the aorta at the same spot, with the same characteristics, likewise associated with considerable narrowing of the coronary arteries, which makes it possible that there was an affection of the cardiac plexus, analogous to that in the first case; but the point, unfortunately, was not investigated.

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[1] Edinburgh Med. Journal. XVI. p. 45. July, 1870.

Analysis of the Symptoms and Special Symptomatology of the Several Forms.

According to the above description, the cardinal symptoms are the pains under the sternum, the feeling of anxiety, the disturbances in the action of the heart and the circulation of the blood, while the changes in the mechanism of respiration are to be regarded merely as consequences of the pain. We shall attempt to refer the first set of phenomena to *definite disturbances of the innervation of the heart, consisting in an abnormal increase or diminution of the activity of the nerves forming the cardiac plexus.*

The *pain* with which the attack begins arises most probably in the nervous plexuses of the heart; this assumption cannot be demonstrated with the same anatomical certainty as in the case of the peripheral nerves whose course is exactly known to us, but one circumstance in favor of it is the fact that the pain always, or nearly always, begins at the spot which corresponds with the site of the heart, and is most acute at the same point. Under normal circumstances, it is true, the heart, like all involuntary organs, is not very sensitive; but we need no more wonder that a stimulation of its sensitive nerves in pathological conditions should excite such extreme pain, than that such should be the case with other organs of vegetable life. The heart derives its sensitive nerves from the vagus, as the experiments of Goltz have shown in the case of the frog's heart, and those of Gurbocki in that of the rabbit's heart. In mammalia other sensitive nerves than those from the vagus seem to enter into the cardiac plexus; for, in spite of the section of both vagi, the animals manifest pain when the auricles are mechanically irritated. Accordingly, we must perhaps ascribe a sensibility to the sympathetic fibres which form so essential a part of the cardiac plexus, and especially so as the character of the pain closely resembles that observed in other irritations of the sympathetic nerve, as in gall-stone colic, in enteralgia, etc.

This neuralgia of the cardiac plexus is sometimes idiopathic, but more commonly it is probably due to mechanical infiltration, pressure, and other injuries of the plexus. Such causes naturally occur to the mind in certain cases of organic disease of the heart, ossification of the coronary arteries, valvular disease of the aorta, and so forth. The cardiac plexus lies behind and below the arch of the aorta and in close proximity to it, so that morbid processes in this vessel may give rise to lesions of the neighboring plexus, as is clearly shown by the results of the autopsy made by Lancereaux. Why these violent attacks of pain occur only at certain periods, in the form of paroxysms, while their supposed cause continues in existence, is no better understood than in the case of other neuralgias.

In those cases where there is no organic change in the heart, we have no idea of the immediate cause of the pain in the cardiac region. Those changes, whether of increase or of diminution of the action of the heart, which occur during the attack, cannot be the sole cause of this violent pain; for the most extreme deviations from the normal action of the heart—both the increase which occurs in aortic defects, and the diminution during fatty change of the muscular tissue—produce a feeling of constriction, but never a pain like that of angina pectoris; while moderate degrees of irregularity in the functions of the heart are often not felt at all.

For this reason I cannot fully agree with Eichwald's theory of the cause of the pain and the nature of the stenocardia. As before stated, he ascribes the attack to arrest of the activity of the heart, due to mechanical obstruction, and explains the pain as a consequence of the exertions made by the heart to overcome the obstruction, since it is found that every over-exertion of a voluntary muscle is painful. Though Eichwald explains the phenomena of the attack very well by means of his theory, yet it is necessary to remark that a change in the action of the heart has not been shown to be the first symptom of a stenocardiac attack; on the contrary, the patient is seized in the midst of the most perfect health, without having had any palpitations. Neither is the action of the heart during the attack by any means so violent as to produce such severe pain. Even if we admit the existence of obstacles to the activity of the heart in certain cases of angina pectoris, yet the heart, in cases of obstacles in the aortic or pulmonary system, usually responds in quite a different way, namely, by increasing the force of its beats. And finally, in those cases of angina pectoris in which the heart, under objective examination, seems quite normal, it is impossible to understand how such hinderances can exist; for the assumption of Eichwald, that irritation of the vagi may constitute such a hinderance, is only tenable when the pulse is retarded. And again, why should a rhythmic retardation of the heart constitute an obstacle to the movement of the blood? We can at any time lessen the rate of the pulse by proper drugs, under pathological circumstances, without ever observing in consequence abnormal sensations. I have seen a pulse of only twenty-eight in the minute, owing to the presence of central disease, without the patient's feeling the slightest abnormal sensation. It is, therefore, doubtful whether a change in the rate of action causes the pain.

In interpreting the *pains which radiate from the præcordial region to other parts of the body, especially the thorax and arm*, we must recollect the anatomical relations of the cardiac plexus and its connections with the nerves of the neck and arm.[1] Those pains which shoot into the region of the *cervical nerves* are explained by the connections between the superior cardiac nerve (from the ganglion cervicale primum) and the anterior branches of the four upper cervical nerves; those extending to the arm, by the numerous connections of the ganglion cervicale medium and inferius (from which the middle and inferior cardiac nerves originate) with the four lower cervical nerves, uniting in the plexus brachialis, and the first dorsal nerve. The fact that the pains are more frequent in the left arm than in the right is perhaps caused in part by the situation of the heart and aorta upon the left side, which naturally gives rise, in case of disease of these organs, to mechanical interference (Zerrung) with the nerves of this side, and in part by the fact that the anastomoses of the nerves are believed to be closer on the left side. The pains in the front surface of the chest are explained by the branches of connection between the dorsal nerves and the brachial plexus. Radiating pains in the region of the diaphragm also occur, which may be referred to the connection of the phrenic nerve with the fourth and fifth pairs of cervical nerves, and through them with the cardiac nerves.

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[1] Compare especially *Lussana*, l. c.

That the pain of angina pectoris is sometimes confined to one spot—the præcordial region, and sometimes shoots out over manifold nerve-paths, is mainly due, apart from mechanical conditions, to the varying intensity of the irritation acting upon the cardiac plexus. According to the analogy of other nervous affections we may assume that in this case the number of nerve-fibres that participate increases with the intensity of the irritation. In several cases, which I had the opportunity to observe for a considerable time, the extension of the pain was always great in proportion to the severity of the initial pain in the cardiac region ; in attacks of moderate severity the shooting to the left arm was almost wholly wanting, the pain reached only to the shoulder, and even the front part of the left half of the chest was affected in a smaller extent of territory.

Certain accompanying symptoms referable to the vagus, as difficult swallowing, vomiting, and difficulty in phonation (Lartigue), are probably also to be regarded as due to irradiation. They must be referred to the numerous connections between the sympathetic and the vagus, especially the share which the latter takes in the formation of the cardiac plexus.

The peculiar feeling of anxiety and oppression seems also to be connected with the pain in the heart; this is, at all events, a more natural assumption than that which derives the feeling of anxiety from an impeded action of the heart. In several visceral neuralgias we find a sensation which quite corresponds with that feeling of oppression, while the latter is entirely wanting in many much severer obstructions to the circulation (as in the various valvular affections).

The *disturbances in the action of the heart and the circula-*

tion, which, in so great a variety of forms, accompany the stenocardiac attack, cannot be supposed to depend upon a single distinct portion of the nervous apparatus, especially in the lack of pathologico-anatomical facts. They must, in accordance with our present knowledge of the physiology of the innervation of the heart and blood-vessels, be referred to several very distinct sources, namely, disturbances in *the automatic, the regulative, and the sympathetic nerves of the heart*, and in the *vaso-motor nervous system*. In this point of view there may be recognized, at least in theory, four possible forms of angina pectoris (as it were, four roots), though the isolated existence of these forms is by no means established with sufficient certainty.

1. *Automatic system of cardiac nerves.* Influences which act directly upon the ganglion masses embedded in the muscle of the heart, such as toxic substances introduced into the ventricular cavity, or solutions into which the entire heart is plunged, are known to be capable of producing immediate annihilation of the contractions of a heart which has been removed from the chest and still pulsates. According to the experiments of Landois,[1] the disturbance of this automatic action of the cardiac ganglia, which takes place under the direct action of certain poisons, may be of two sorts: if weak solutions are injected into the endocardium of the frog a stimulation of the ganglion cells occurs, with an increase in the rate of speed of the heart; but if the solutions are stronger, the ganglia are rapidly paralyzed and the heart is arrested. In a similar way the ganglia of the heart may be affected in pathological conditions. If their rhythmic activity is interfered with in any way, either by the existence of abnormal resistance to the movement of the blood, as in aortic disease and atheromatous processes; or if the ganglion receives too little blood, as in contraction and closure of the coronary arteries; or if the latter are affected in conjunction with the muscular tissue of the heart, in myocarditis or fatty degeneration—in any of these cases the action of the heart may be changed in one of two ways: either the rapidity of its movements is *increased*, if the above pathological influences produce an *irritation* of the

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[1] Die directe Herzreizung. Greifswalder med. Beiträge. II. 1864. S. 161.

ganglia, or it is *diminished*, if the influence produced be a *paralyzing one*. The assumption, therefore, that in a stenocardiac attack the automatic ganglia are disturbed in their activity, may agree very well with the fact that the action of the heart during the attack is sometimes increased, sometimes diminished. The form of angina pectoris, accompanied by disturbance of the automatic ganglia, might be denominated the *cardial excito-motor* form, or the *cardiocentric ganglious* form.

In favor of the theory of an involvement of the ganglia of the heart—perhaps due to a deficient supply of blood, as in case of disease of the aorta and contraction of the coronary arteries—another experimental fact may be adduced. Von Bezold[1] observed in every case a change in the beats of the heart, when he cut the vagi, cervical sympathetics, and the cervical medulla, and then closed the large coronary arteries or several of their branches with forceps. After ten or fifteen seconds the beats became less frequent, then irregular, with alternations of slow and quick contractions, and in a minute or a minute and a half the ventricle was entirely relaxed. If the closure of the vessels was suspended, the pulsations began afresh and soon became quite regular.

2. *Regulative system* of cardiac nerves. In the majority of cases of angina pectoris the rapidity of the pulse is increased; if, therefore, the vagus were affected, we should have to infer that it was in a state of temporary paresis, causing a diminution of its regulative action upon the heart. But some cases occur, with *retardation* of the pulse, and even with a *temporary arrest* of the heart, which are probably due to a state of irritation in the vagus, which therefore might be called the *regulative* form of angina pectoris. Under this head must be placed the case related by Heine (see above), in which the autopsy revealed, among other things, changes in the vagus; also two cases by Canstatt, in which a temporary arrest of the action of the heart, with a feeling of great anxiety, formed the chief symptoms.

Eichwald also mentions a case in which a hysterical attack, whenever it occurred, was always accompanied by a stenocardiac paroxysm, during which the pulse was retarded, with increased force in the single beats; the pulse was hard and full, but at the same time the beats were few and slow. If the paroxysm lasted somewhat longer, the pulse became irregular, intermitted, and even became imperceptible for whole minutes. The action of the heart was of a similar character in two other cases of angina pectoris, in one of which the patient was hysterical, and in the other a pneumonia had produced a great loss of strength, and the paroxysm was provoked by a violent mental agitation. Eichwald therefore thinks that in such cases there is a state of irritation in the region of the vagus; in favor of this supposition is the observation that in such cases other symptoms referable to the vagus occur, as difficulty of phonation and of swallowing, and that the change in the action of the heart in this kind of angina pectoris is closely related to the experimental results obtained in irritating the vagus. For in slight irritation of the vagus the beat of the heart, as in the beginning of a stenocardiac attack, becomes slower and stronger; if the irritation is greater, as in an attack of longer duration, the action of the heart is retarded, and even more or less stopped. The paroxysm of pain is also very well reconcilable with the theory of irritation of the vagus, since the sensibility of the heart depends chiefly upon the ramifications of the vagus.

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[1] Centralblatt 1867, No. 23.—Compare also *Suchtschinsky*, ibid. 1868. No. 3.

Besides the cases of angina pectoris that are due to direct irritation of the vagus, there are certain others which occur with especial frequency in diseases of the abdominal organs, which physiological facts allow us to consider as *reflex vagus-neuroses* (according to Landois, *angina pectoris reflectoria*). It is known that the heart can be brought to a stop in diastole by irritation of the sympathetic fibres in the abdomen, just as by direct irritation of the vagus; there must, therefore, be fibres of the sympathetic which enter the spinal cord by the rami communicantes, and, passing upward through the dorsal to the cervical portion, there transfer their impression of irritation to the centre of origin of the vagus; for if both vagi have been cut, or the medulla oblongata destroyed, the irritation of the sympathetic has no longer any effect upon the heart. Cases reported in older literature, in which disease of the abdominal viscera was accompanied by stenocardiac attacks, are found in the monograph of Ull perger. From later literature I will mention a case of ang pectoris, in which a course of treatment directed toward the li which was in an enlarged condition, immediately produced amelioration of the general symptoms;[1] the stenocardiac attac which previously occurred whenever violent bodily motion v indulged in, disappeared entirely. There was no affection of heart in this patient.

3. *Sympathetic cardiac system.* The accelerator nerves the heart, running in the course of the sympathetic, dem strated by von Bezold, permit us to refer certain cases of ang pectoris, with accelerated action of the heart, to an increase the activity of the rhythmically-acting cardiac ganglia, due the influence of the sympathetic. We might designate this fo as *sympathetic excito-motor* angina pectoris, in contradistinct to that caused by a direct affection of the automatic card ganglia. Since all the sympathetic fibres of the heart, fr whatever source arising, are united in the cardiac plexus, the ter furnishes a specially probable source of abnormal activity the heart; and this assumption is notably supported by autopsies reported by Lancereaux.

4. *Vaso-motor nervous system.* In disturbances of the inn vation of the vaso-motor nerves (which likewise run with sympathetic, for the most part), a *change in the tone of the v sels* must occur, and in consequence a *change of blood-pressu* which may react upon the heart in two ways: if these ner are irritated, the result will be a contraction of the vessels; they are in a paretic condition, the vessels will be dilated. the former case the contracted condition of the peripheral cir lation creates an obstacle to the emptying of the heart, a therefore the blood-pressure rises throughout the aortic syst with an increase of force in the action of the heart; while in latter the resistance is lessened, the aortic pressure diminish and the heart works with less force.

This is the class of cases for which Landois has offered very suitable name of "*angina pectoris vasomotoria.*" W them belong those described by Nothnagel, in which, the heart being quite healthy, attacks of angina pectoris occurred under the form of general arterial spasm, often produced by the influence of cold. After some premonitory symptoms of abnormal sensation in the extremities, as numbness, the feeling of cold, and so forth, a sensation of anxiety and palpitation followed, and even a dull pain, beginning at the heart and spreading into the left thoracic region. The objective phenomena corresponding to these symptoms were pallor and loss of temperature in the skin, diminution of the sensibility of the skin, cyanotic discoloration of the extremities; the radial artery was sometimes a little smaller than normal, the sounds of the heart clear, the action of the heart rhythmical and sometimes increased in force, the number of beats unchanged. The attacks were relieved by treatment suited to relieve vascular spasm, by such remedies—especially irritants and warmth—as increase the flow of blood to the skin. Nothnagel explains the palpitation in these cases as due to the resistance to the action of the heart presented by the extensive contraction of the circulatory apparatus; the feeling of constriction and pain in the region of the heart he takes to be due to the excessive exertion of the heart, thus assuming an explanation like that of Eichwald.

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[1] *Bergson*, Deutsche Klinik. 1862. p. 48.

Whether, in addition to the vaso-motor nerves, the depressor nerve discovered by Ludwig and Cyon, which seems to act as a regulator of the variations in blood-pressure, can assist in causing the disturbances of circulation in the stenocardiac attacks, is a question which cannot at present be judged.

It follows that we have these types, namely:

1. *Excito-motor cardiac* or *cardio-centric, ganglionic angina pectoris*, from direct lesion of the automatic excito-motor ganglia of the heart; it may assume the form of irritation (with increased rapidity of pulse), or of paralysis (with retardation of pulse).

2. *Regulator angina pectoris*, from lesion of the cardiac system of nerves of arrest (vagus). *a. Direct neurosis of the vagus*, either in the form of irritation (retarded pulse and increase in force of pulsations of heart, full, hard pulse, with disturbance of phonation and deglutition; sometimes a temporary arrest of the heart), or, more rarely, in the form of paralysis (acceleration of

pulse). *b. Reflex neurosis of the vagus* (angina pectoris reflectoria), originating in disease of the abdominal organs, with the symptoms of irritation of the vagus.

3. *Excito-motor sympathetic angina pectoris*, from lesion of the accelerator nerves of the heart which run with the sympathetic. (Symptoms as in the first form.)

4. *Vaso-motor angina pectoris*, from affection of the vaso-motor nerves, running in the sympathetic for the most part—either in the form of irritation (contraction of the vessels, increased pressure, with normal or but slightly increased frequency of pulse; symptoms of arterial anæmia, paleness and coldness of the skin, etc.); or, more rarely, in the form of paralysis, with the opposite set of symptoms. Of course, the complication of these principal types and their confusion with each other are not at all impossible. The great variability of the circulatory symptoms accompanying the attack is thus paralleled in the variety of the pathological conditions.

Diagnosis and Prognosis.

The pathognomonic symptoms of the attack are such as hardly permit confusion in the diagnosis of angina pectoris; but this is not a very great gain. The practically important thing to know is whether there be organic disease of the heart or vessels, or whether the disease be a pure neurosis; and, if the latter, what is the special form of disease, or (which is the same thing) what is its point of origin in the several nervous regions. This problem will often be hard of solution, especially as the physician seldom is in a position to view the attacks, and to apply the proper corrections to the descriptions given by the patient or his friends.

The prognosis of angina pectoris depends first upon the question whether or no there are complicated diseases of the vascular apparatus (valvular disease, fatty degeneration, sclerosis of the arteries). If such exist, the prognosis is naturally bad, in proportion to the severity of the fundamental lesion. In purely nervous angina pectoris, the prognosis, as concerning life, is favorable upon the whole, for grave sequelæ and death are rarely caused by the stenocardiac attacks, however severe they may seem. Some cases are, indeed, reported in which death is said to have occurred in the course of the attack; but these seem—and Lancereaux's autopsies support this view—to be cases in which the aorta and the coronary arteries were greatly altered, with consecutive changes in the cardiac plexus, and they, therefore, ought not to be classed as purely nervous. This differentiation, it is true, will often be without value in actual practice, for the special diagnosis cannot be sufficiently relied upon, and therefore, if the attacks are at all of a nature to arouse our apprehension, we ought to be fully warned against too sanguine hopes. As regards the disease, the prognosis is certainly quite unfavorable, as a radical cure, by nature or by art, is observed in an extremely minute proportion of the cases. It is most favorable in cases which, without constitutional predisposition, can be traced to such proved evil influences as tobacco-smoking, exposure to cold, etc., and usually take the form of angina pectoris vasomotoria. In those cases, also, which appear under the form of reflex neuroses of the vagus, there may be hopes of a cure after the abdominal disease is relieved, provided the latter can be accomplished.

Treatment.

In cases in which physical examination shows the presence of valvular disease or fatty degeneration of the heart, etc., or in which the various senile changes, peripheral arterio-sclerosis, etc., permit us to infer atheromatous disease of the aorta and the coronary arteries, the treatment of angina pectoris is essentially the same as that of the fundamental lesion. In cases, also, where other causes, such as abdominal disease or excessive tobacco-smoking, are found, we shall begin the treatment of angina pectoris by removing these. Commonly, however, the causes of the affection are unknown or obscure, and the causal treatment is, therefore, out of the question in most cases.

We possess, however, as in the case of all diseases of which the etiology and pathogenesis are enveloped in a natural or artificial obscurity, a goodly number of remedies, empiric, symptomatic, or so-called specific, most of which are wholly useless, while the rest confine their action to palliation, or produce a radial cure only by way of exception.

The general and local blood-lettings during the attack, formerly so much praised, will hardly be thought of at present by any one; while, on the other hand, all sorts of so-called derivative remedies, as frictions, mustard poultices, stimulating baths for the feet and hands, stimulating embrocations, and so forth, are still very popular. It is certain that the latter are more useful than the internal administration of the so-called nervines and antispasmodics, valerian, musk, castoreum, camphor, succinate of ammonia, and the like, as well as of the narcotics. Other things recommended in the attack are the application of cold, and in some cases of warmth; inhalations of ether or chloroform in small doses, not sufficient to produce full narcosis (Romberg); narcotic clysters; endermic and hypodermic administration of narcotics. As regards the latter, hypodermic injections of morphia doubtless often exercise a palliative action during the attack, not only upon the pain, but also on the associated disturbances of circulation. This may perhaps be ascribed, for the most part, to the paralyzing action of large doses of morphine upon the vaso-motor nervous system (Wolff, Mendel). I have observed very decided symptomatic benefit of this kind from morphine injections, not only in uncomplicated (vaso-motor) angina pectoris, but also in a case accompanied by an affection of the valves (insufficiency of the aortic valves, with stenosis at the ostium venosum sinistrum and hypertrophy *en masse*). I have seen slighter benefit resulting from atropin and from coniin (recommended by Erlenmeyer) when injected subcutaneously. In those well-defined cases characterized by violent accelerated action of the heart, due to abnormal irritation of the automatic ganglia or the excito-motor sympathetic fibres, the class of special "heart poisons,"[1] such as atropin, nicotin, coniin, aconitin, delphinin, veratrin, physostigmin, etc., may yet be found applicable.

The *nitrite of amyl* is a remedy which, in many cases, is justified both upon rational grounds and by its power to relieve symptoms. Brunton, arguing from the fact that the arterial tension increases in the stenocardiac attack, has recommended the inhalation of this remedy, the effect of which, as was stated in reference to hemicrania, is to produce a dilatation of the capillaries, probably through peripheral palsy of the vaso-motor nerves. Leishman, Sanderson and Anstie, and Wood, like Brunton, found the results beneficial; while Fagge found the remedy inactive. Probably the favorable effect may be confined to cases possessing the character of vascular spasm. As it so easily causes syncope, it should if possible be used with even greater care than in cases of hemicrania.

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[1] Cf. *Boehm*, Studien über Herzgifte. Würzburg, 1871.

The general treatment of this disease is more a puzzle than even the symptomatic; the remedies are many, the cures few. Various metallic preparations have been much praised; the forms of iron, sulphate of zinc (Perkins), cyanide of zinc (Copland), nitrate of silver (favorably mentioned by Romberg and others in hysterical angina pectoris), and arsenic (lately recommended in high terms by Lebert, Cahen, and Philipp). I have tried the latter without any important benefit in several cases of uncomplicated angina pectoris. I have also used the bromide of potassium, as well as the bromide of calcium, which Hammond[1] proposes for a substitute, but without permanent effect; the latter in quantities up to five grammes per diem. Others have strongly recommended quinine, phosphoric acid (Baumes), inhalations of oxygen (Kneeland), digitalis, hydrocyanic acid, and, in view of the supposed arthritic origin of the disorder, those remedies called antiarthritic. Laënnec recommended the wearing of a magnetic plate. Somewhat more regard must perhaps be given to the so-called derivation by the application of irritating plasters, issues and setons in the cardiac region. Koehler, Wittmaack, and others have obtained a cure by this method in a few obstinate cases, after the failure of most of the usual remedies. The induced current, in the form of the faradic brush, seems to act in a similar way. Duchenne[2] affirms that he has not only arrested very severe attacks instantly and completely by cutaneous faradization of the nipple and region of the breast, but that he has, by protracted use of this means, produced a permanent cure in two cases, one of the uncomplicated nervous form and one of the hysterical form.

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[1] New York Med. Journal. 1872.

[2] Électrisation localisée. 2d. ed. p. 967.

Any treatment, or rather any attempt to treat, that claims to be based upon reason, will in future have to distinguish between the several forms of angina pectoris more closely than has usually been done. *Cutaneous irritation*, and especially cutaneous faradization, which acts most quickly, may perhaps have a great part to play in certain forms of the disease, owing to its power of acting reflexly upon the nerves of the heart and the vaso-motor nervous system. This action is, of course, of two opposite kinds, in the case of weak and of strong irritations. The former strengthen the contractions of the heart, accelerate the circulation, and contract the vessels, by reflex excitation of the excito-motor and vaso-motor nervous system; but the latter weaken the contractions of the heart, retard the circulation, and dilate the vessels, by reflex excitation of the regulator nerves of the heart and paralysis of the vaso-motor centres. In analyzing the symptoms we have seen that the disturbances of the action of the heart and the circulation of the blood in angina pectoris usually possess the character, either of an irritation of the excito-motor nerves of the heart, or of irritation of the vagus, or of increased excitement of the vaso-motor system of nerves (angina pectoris vasomotoria). Of these three types, more or less clearly defined, only the first and the last can call for or justify the employment of severe cutaneous irritation; that is, those cases which are marked by a powerful, rapid, violent action of the heart, arteries contracted to a cord, small, tense pulse, etc. But when the symptoms of irritation of the vagus and of vascular paralysis are most prominent, or when, during the attack, the symptoms very soon revert to this type, then cutaneous irritation must not be used at all, or only in its weakest form.

The therapeutic use of the constant current must be founded upon a similar consideration of the symptoms of each special case. If correctly employed, it is probably a remedy of chief importance, and perhaps the only direct remedy for angina pectoris. But the nature of the symptoms will direct us when to select methods of application which produce reflex excitation of the regulator nerves of the heart, and when to prefer direct galvanization of the cervical sympathetic and cervical vagus. I have only been able to use the former procedure—in three cases of accelerated action of the heart without organic disease—but could not continue the application long in either instance. The effect was distinctly good ; the attacks became less severe ; and in one case ceased entirely, while previously they had made their appearance almost daily. In a fourth case, recently brought under treatment, the attacks have become rarer and milder. The method used consists in the application of strong stabile currents, rising to the number of 30 elements; the positive pole, with a broad surface, is placed upon the sternum, while the negative is placed on the lower cervical vertebræ. Von Huebner has lately obtained a permanent cure by similar methods in a case which seemed to have a rheumatic origin. He placed the positive electrode upon the fossa suprasternalis, and the negative upon the cervical ganglia of the sympathetic, on both sides in succession ; then he placed the positive pole upon the lowest cervical ganglion, and the negative upon the sensitive spots, at the angles of both shoulder-blades. At first only very weak currents, of from 4 to 6 elements, were borne. The attacks ceased from the first session, and did not return ; and by degrees it became possible to use stronger currents, of from 8 to 10 elements.

UNILATERAL PROGRESSIVE ATROPHY OF THE FACE.

(*Hemiatrophia facialis progressiva.*)

Parry, Collections from the unpublished writings. I. p. 478.—*Romberg*, Klinische Ergebnisse. Berlin, 1846. p. 75, and Klinische Wahrnehmungen und Beobachtungen. Berlin, 1851. p. 83.—*Stilling*, Physiologische, pathologische und medicinisch-praktische Untersuchungen über die Spinal-Irritation. Leipzig, 1840. p. 325.—*Bergson*, De prosopodysmorphia sive nova atrophiae facialis specie. Diss. inaug. Berlin, 1837.—*Hueter*, Singularis cujusdam atrophiae species nonnulli. Diss. Marburg, 1848.—*Schott*, Atrophia singularium partium corporis quae sine causa cognita apparet trophoneurosis. Diss. Marburg, 1851.—*Moore*, Dublin Journal. 1852.—*Samuel*, Die trophischen Nerven. Leipzig, 1860.—*Eulenburg* und *Landois*, Die vasomotorischen Neurosen. Wiener med. Wochenschrift. 1866.—*Hering*, Archiv f. klin. Chirurgie. IX. 1. 1867.—*Baerwinkel*, Beitrag zur Lehre von den neurotischen Gesichtsatrophien. Archiv der Heilkunde. 1868. IX. p. 151.—*Guttmann*, Ueber einseitige Gesichtsatrophie durch den Einfluss trophischer Nerven. Archiv f. Psychiatrie. 1868. I. p. 173.—*Panas*, Session of the Société de Chirurgie. May 5, 1869.—*M. Meyer*, Sitzung der Berlin med. Gesellschaft. Nov. 17, 1869.—*Hitzig*, Sitzung der Berl. med. Gesellschaft. Dec. 1, 1869.—*Lande*, Essai sur l'aplasie lamineuse progressive (atrophie du tissu connectif), celle de la face en particulier. Paris, 1870.—*Eulenburg*, Lehrbuch der functionellen Nervenkrankheiten, 1871.—*Seeligmueller*, Ueber Sympathicus-Affectionen bei Verletzung des Plexus brachialis. Berl. klin. Wochenschrift. 1870. No. 26, and 1872, No. 4.—*Brunner*. Zur Casuistik der Pathologie des Sympathicus. Petersb. med. Zeitschr. N. F. II. 1871. p. 260.—*Tanturri*, Emiatrofia facciale progressiva e trofonevrosi di Romberg. Il Morgagni. 1872. No. 11. 12.—*Frèmy*, Étude critique de la trophonévrose faciale. Paris, 1873. —*Baerwinkel*, Neuropathologische Beiträge. Deutsches Archiv f. klin. Med. XII. p. 606.—*Nicati*, La paralysie du nerf sympathique cervical, étude clinique. Lausanne, 1873.—*Charcot*, Leçons sur les maladies du système nerveux. 2ème édit. Paris. 1875.

This extremely rare disease is characterized by a *chronic loss of substance in one side of the face, which usually begins in the external soft parts and passes successively to the deeper tissues;* the neurotic nature of its origin is not proved by pathological anatomy, but is rendered very probable by internal and external reasons.

History.

The first case of this sort seems to have been described by Parry in 1825. Bergson, in 1837, described a case taken from Romberg's policlinic, and pointed out the possibility of the implication of the nerves of the vessels. Stilling, in his very interesting work on Spinal Irritation, to which far too little attention has been paid, and which promises in its title much less than it actually contains, mentioned a case observed by Schuchardt, and explained the atrophy as due to a functional disturbance of the vascular nerves, especially those which run in the trigeminus to the vessels of the head. His hypothesis that this was a case of "diminished reflexion of the sensitive vascular nerves upon the corresponding vaso-motor nerves" is certainly somewhat forced. The disease was more exactly described by Romberg (1846 and 1851), who conceived it to be a primary trophoneurosis—an assumption thoroughly established by Samuel in his pioneer work upon the trophic nerves. Axmann-Hueter, Moore, Baerwinkel, Guttmann, M. Meyer, Hitzig, Bitot-Lande, Brunner, Tanturri, and others, have added to our knowledge of the disease by separate observations, as yet very few in number. The disease was considered theoretically by Moore as a distinct form of progressive muscular atrophy in the region of the seventh pair of nerves, while Baerwinkel referred it to a disease of the Gasserian or the spheno-palatine ganglion, Brunner to a permanent state of irritation in the cervical sympathetic, and Lande quite denied its neurotic character and preferred to assume a genuine primary atrophy of the adipose tissue. The discussion upon these points is still open.

In the first accounts no special name was given to the disease, while Romberg described it simply as "trophoneurosis," and Bergson more specifically as "prosopodysmorphia." Later authors chose the terms "neurotic atrophy of the face" (Samuel, Baerwinkel), or facial trophoneurosis. More lately Lande has proposed the name "aplasie lamineuse progressive" or "atrophie du tissu conjonctif," which, however, presupposes that the atrophy is confined to certain tissues, a supposition by no means justified by the results of examination in most cases, especially advanced ones. I consider the designation "hemiatrophia facialis progressiva" as the completest and most suitable, since the essential features of the disease, as established by all previous investigations, consist in its limitation to one side and its tendency to progress. The term seems to me also preferable to that of "neurotic atrophy of the face," as the latter is based upon a hypothesis regarding the origin of the disease—a very probable one, but not yet demonstrated.

Etiology.

As regards predisposing influences no hereditary transmission of the disease has as yet been observed. But it is certain that *youth* and the *female sex* are predisposing circumstances. Out of 16 reported cases which may with certainty be classed under this disease, 5 were observed in men, 11 in women. In all the cases the disease commenced before the twenty-fifth year of life, namely, at the ages of 2, 3, 6, 7, 10, once each; at 11 three times; at 12 once, 13 once, 15 twice; at 18, 22, 23 and 24 once each.

It is noteworthy that the disease has a special predilection for the left side of the face; among sixteen cases, thirteen were upon the left and only three on the right side.

In a few cases the outbreak was preceded by scarlatina (Bergson), measles (Hueter), whooping-cough and a local herpetic eruption (Schuchardt). As occasional causes, a severe cold taken by exposure to draft and the like are sometimes mentioned, sometimes a traumatic lesion; Schuchardt, for example, speaks of a fall on the head, which left a cicatrix on the right parietal bone, below the coronary suture. In several cases the commencement of the atrophy has been preceded for a longer or shorter time by notable disturbances of innervation, especially symptoms of sensory or motor irritation. Tearing pains in the head, and pains in the corresponding side of the forehead or superior maxillary region, are frequently mentioned; also, toothache (Baerwinkel). In one case (M. Meyer) the patient had suffered from epileptic fits for several years previously; of these fits there were two sorts: one severe, lasting several hours, and separated by long intervals; the other slight and more frequent, in which the side of the face which afterwards atrophied was the only part much affected. In Brunner's case also, epileptic attacks, occurring for the first time during pregnancy, continued for a year before the outbreak of the disease. In Parry's case a (hysterical?) left hemiplegia preceded the disease, with transitory disturbance of intelligence, but these had passed away two years before the beginning of the disease. In a case observed by Axmann and Hueter, the patient, a journeyman weaver, aged thirty-two, had suffered from irregular spastic contractions of the masticator muscles of the left side, which commenced in the seventh year of his age, and began to grow less frequent from his fourteenth year, but had never quite left him. These spasms were associated with an increased delicacy of sensation in the region of the left trigeminus, especially the first and second branches, differences of temperature being noted with much greater clearness and sharpness than on the right side. The atrophy developed itself not long after the appearance of these phenomena; it is remarkable that it was chiefly confined to the region supplied by the third branch, namely, the temporal and inferior maxillary region.

Symptoms and Course.

The first and most striking symptom, in a certain number of cases, consists in a peculiar spotty *discoloration of the skin*, associated with a thinning and emaciation of the latter. A white spot appears on the face, and gradually spreads; the spot, pale or perfectly white at first, may afterwards get a yellowish or brownish tint, as is often observed in cicatrices after burns. Sometimes several such white spots are formed at once or successively, and afterwards run together to one spot of considerable extent. The discolored spots soon become the seat of a marked atrophy, which comes to view either about the same time with the discoloration, or some time later. The skin seems sunken at these places, and as the disease goes on, it becomes deformed by pits of greater or less size and depth. These are manifestly caused by the *loss of subcutaneous fatty tissue*, which proceeds so far that, in places where there was formerly an abundance of fat, the skin lies directly upon the bones, can be raised with difficulty, and when raised its folds are often as thin as two millimetres. It is, however, certain that the proper tissue-elements of the cutis, and even the epidermoid structures, take part in the disease; this is shown by the frequency with which the nutrition of the hair, the secretions of the skin, and other functions are disturbed. The change in the structure of the *hair of the beard*, the *eyelashes and brows*, and even the *hair of the head* on the same side, may precede the formation of the above-mentioned spots and pits; neuralgic sensations may be, or may not be, associated with it. The hair sometimes loses its color and turns perfectly white; it sometimes falls out, or its growth is more or less interfered with. Sometimes only a few streaks of the hair of the head or of the lids and brows are discolored.

The *cutaneous secretion* is in most cases much diminished or wholly stopped on the atrophied side. This is especially true of the secretions of the sebaceous follicles, while the sweat-glands often act in a normal manner. The contractility of the smooth muscular fibres of the skin, as, for instance, under electrical stimulation, remains unaltered. In more advanced stages the atrophied skin often feels rough, or even cicatricial, and sometimes desquamates rapidly. The cutaneous sensibility has rarely been found lessened (only by Tanturri). In some cases, on the contrary, an increased susceptibility to external stimuli, as that of electricity, is said to have been remarked in the affected portions of the skin. But subjective sensations, paralgic or neuralgic phenomena in the corresponding regions, have often been conjoined with the loss of color and the disappearance of the hair. Besides these precursory neuralgias of the trigeminus, especially the supraorbital branch, neuralgic attacks have been observed during the development and progress of several cases. In other instances no painful sensation has been felt in the affected side of the face, either before or during the progress of the disease, so that pain is, at all events, not a necessary and pathognomonic symptom of the morbid process. In two cases, observed by Lande in Bitot's wards, the patients complained of a continual feeling of itching in the skin, and of constriction as if a rubber mask were applied to the seat of atrophy.

The *deeper tissues* are affected differently and very unequally by the impaired nutrition, but it is necessary to remember that it is very hard to make a just or even an approximate estimate of the degree of implication in regard to them.

The *muscles* of the affected side appeared in most cases quite unaffected, even after the process had run on for years; no lessening of their volume was observed; they contracted with their normal energy and reacted to the electric stimulus just like the muscles of the well side. In other cases their volume was lessened, and sometimes fibrillary twitchings have been observed. The face often seemed to be somewhat drawn towards the atrophied side. In the case examined by Guttmann and myself a distinct emaciation and atrophy could be seen in the muscles of mastication innervated by the trigeminus—namely, the masseter and temporalis. The movements of mastication were also weaker upon the atrophied side, and the electric contractions were less energetic. In all the muscles, however, which received their nervous supply from the facialis, there was no visible want of symmetry. In several cases, however, this want appeared in the muscles of the upper lip; not only the skin, but also the portion of lip covered by mucous membrane, was decidedly thinner on the atrophied side than on the other, so that only a small strip of red was to be seen, and when the mouth was partly opened an oval aperture was formed before the lips had parted on the other side (Hueter, Bitot and Lande, Hitzig). Since the corresponding part of the upper lip is mainly composed of fibres of the orbicularis oris, a partial atrophy of this muscle must be supposed to exist.

The larger *blood-vessels*, so far as they were accessible to direct examination, seldom showed any distinct changes. The large arteries of the face, especially—maxillaris externa, temporalis, etc.—appeared unaltered in calibre, in most cases. In some cases they are said to have been smaller than those on the well side. The atrophy laid the arteries and veins open to sight and touch, and this circumstance may readily have given rise to mistakes regarding their calibre.

The tone of the small arteries seems, as a rule, to be retained or even increased. For, while the atrophied parts are usually pale or quite white, they are still capable of blushing under mental influences, under excitement or in exertion. A sudden suspension or diminution of the tonic contraction must therefore be supposed to take place in this case. Local electrical stimulation (cutaneous faradization and galvanization) also will often redden the pale spot. But in some cases the power to turn red in response to psychical influence is lost, so that when the well side blushes the atrophied side remains white ; in such cases the electric stimulus is also without effect. But the power to blush may return, without any improvement taking place in the volume and coloration of the parts. These circumstances show us at least that distinct changes in the vascular tone are not necessarily connected with the atrophy. The temperature of the skin on both sides of the face also feels the same ; and the thermometer, applied externally, in the mouth or in the external auditory canal, shows no difference.

The *bones of the face* in some cases have been found by exact measurement to be decidedly atrophied, and in parts very much so. This is true of both the lower and the upper jaw, and the smaller facial bones connected with the latter (malar bone). The *cartilages, e. g.*, those of the nose, lose in size in the course of time. In one case there is said to have been an abnormal laxness and dryness in the articulation of the jaw on the affected side. The teeth may experience consecutive alterations in consequence of atrophy of the upper and lower jaw ; as the jaw retreats, the teeth push each other out of place, and the upper set interferes with the lower. In one case, that of a child, an incisor tooth was missing on the atrophied side, and its next neighbor was very imperfectly developed.

Of the organs in the interior of the mouth, the *tongue* may be diminished in size on the affected side, which is said to be the reason why it deviates towards this side when put out ; but the latter circumstance is much more likely to be due to atrophy of the outer parts and the upper lip. The *vault of the palate*, the *soft palate*, and the *uvula* may also take part in the atrophy. The secretion of saliva and the movements of swallowing were not interfered with in any case; but in one case, in which the external atrophy had reached as far as the region of the larynx, the pronunciation of the letter *r* was somewhat impeded.

The functions of taste, smell, hearing, and seeing were not interfered with in any of the cases reported; in one (Bitot), in which the hearing was affected, this was owing to accidental and unessential complications. The secretion of tears is normal. The orbital fatty tissue behind the globe of the eye often disappears at the same time with that of the face, which makes the eye upon the atrophied side appear sunken and smaller, and the opening of the lids narrower.

In Brunner's case, which is peculiar in several other respects, the *opening of the lids was wider and the eyeball more prominent; the pupil was at the same time dilated and sluggish in reaction.* The conjunctiva was pale and deficient in blood; the secretion of tears and mucus diminished. The outer ear of the affected (left) side was much thinner, smaller and colder than that of the right; the temperature in the auditory meatus lower by nearly 1°, in the left side of the mouth by 0.2° C. *Pressure upon the ganglion cervicale supremum gave pain upon the left side*, but not on the right. The rate of the pulse varied from 88 to 100 in the minute.

The disease is always slow and very protracted. The cases hitherto reported have been under observation from three to twenty-three years. In most cases the disease marches steadily and regularly forward; in some a brief pause seems to occur which very soon gives way to a new onset of the disease. Whether at a certain point of its progress a permanent arrest occurs, cannot yet be determined; but a few cases (Tanturri, Baerwinkel) appear to bear testimony to this, and even to permit the possibility of a spontaneous improvement. No extension beyond the affected half of the head has been observed as yet. The general health of the patient is not at all interfered with by the disease; he often enjoys perfect health, except there be some complications, such as epilepsy.

Analysis of the Symptoms.

The historical account has already shown how great a variety of theories have been set up to explain the symptoms. In general we can distinguish between theories which assume a neurotic origin for the disease and those which regard the atrophy as idiopathic, and independent of disturbances of nutrition. The neurotic theories differ among themselves chiefly in respect to the point whether the *vaso-motor* or the *trophic nerves* are primarily affected; whether the disease is to be regarded as an angioneurosis or a trophoneurosis; and with this question is associated the one regarding the implication, exclusively or chiefly, of various nerves of the head, especially the *facialis*, the *trigeminus* and its ganglia, and the *cervical sympathetic.* It will presently appear with how little certainty we are able to answer these questions. The absolute want of data from pathological anatomy throws us back upon the data of physiology and experiment, which, beautiful and abundant as they are, yet present but a dim and confused, often suspicious analogy with the phenomena of the disease. I will state at once that, in my view, it is not probable that all the cases observed can be explained by one and the same scheme, but that some cases show most clearly a neurotic origin (sometimes certain branches of the trigeminus being primarily affected, sometimes the cervical sympathetic), while in other cases a neurotic origin cannot be quite denied, but neither can it be maintained by distinct evidence.

The necessity of establishing such a distinction may be better seen by an analysis of certain cases, which are especially instructive from the pathogenetic point of view.

Vaso-motor and Trophic Theory.

Bergson, who was the first to speak of the possibility of a primary affection of the vascular nerves, supported his view by the fact that in the case described by him the carotid of the left, or atrophied side, pulsated less strongly than that of the right. This symptom is not only too indefinite, but it was absent in most of the cases. Stilling, who attempted to formulate more exactly the degree of implication of the vaso-motor nerves, supported his view by the case of Schuchardt, which has already been mentioned repeatedly. This was the case of a girl of twenty-six, the right side of whose face had been undergoing a process of slow atrophy ever since her third year, and was r extremely wasted; the cause assigned was a fall from her nurse's arm, which had left a scar on the right temporal bone below the coronary suture. Stilling explains the atrophy by "a diminished reflexion of the sensitive nerves of the vessels upon the corresponding vaso-motors," the sensitive nerves intended being the branches of the second division of the trigeminus, distributed upon the arteries of the face; the cause was possibly a circumscribed lesion of those fibres, due to shock, laceration or extravasation, occurring at the time of the fall. But it is hard to see why we need assume a lessened reflexion from sensitive to vaso-motor fibres, and not rather a direct lesion of the latter as a cause of atrophy, since the functions of sensation and sense in the region of the trigeminus were in no respect altered.

Romberg, as already related, had designated the disease as a trophoneurosis, without explaining himself precisely as concerning its origin. Samuel developed more fully the relation of the disease to the trophic nervous system. He justly urges that a lesion of the vascular nerves, with a resulting diminution or stopping of the circulation of the blood, cannot be considered as a cause of the disease. For in such cases the result would probably be inflammation, softening, gangrene, and never simple atrophy; or a collateral circulation would be established, and with it the nutrition would again be rendered normal. Neither irritation nor paralysis of the vaso-motor nerves, experimentally practised, gives rise to the symptoms of simple progressive atrophy of all or most of the tissues, such as exists in the present disease. Samuel therefore considers that the lesion must concern certain trophic nerves, the paralysis of which causes a falling out of the hair and the nails of the fingers and toes, besides other symptoms, the like of which has been observed in experimental sections of nerves performed upon animals.

The interesting experiments of Mantegazza,[1] who followed out in detail the histological changes of the several tissues after section of nerves, have shown that the deeper tissues (muscles, bones, periosteum, connective tissue, lymphatic glands, etc.) take part in the consecutive alterations; for instance, the muscles become atrophied, and the bones experience a great change of nutrition, which is always connected with loss of weight. The muscular atrophy which is associated with interstitial growth of connective tissue ("muscular cirrhosis") has also been confirmed by Vulpian,[1] Ziemssen, Weiss, and Erb, in their well-known experiments. After experimental sections of nerves we observe not only atrophy, but also hypertrophic and hyperplastic alterations. Mantegazza observed hyperplasia of the connective tissue and periosteum, hypertrophy of the medulla, osteophytic formations, and hypertrophy of the lymphatic glands to the extent of sixfold. A somewhat isolated case, related by Stilling, may perhaps be brought into connection with these facts; in this instance an injury suffered by a nerve was followed, not by atrophy, but by *hypertrophy* of one whole half of the face in connection with disturbances of sensibility and motility in the region of the trigeminus.

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[1] Giornale Veneto di scienze mediche. ser. 3. tom. 6. 1867.

The much discussed question of the existence or non-existence of the trophic nerves cannot be entered upon in this place. Although of late the hypothesis of trophic nerves has been regarded as needless or as unproved by many (Lande, Onimus), yet the majority of authors are strongly inclined to answer the question in the affirmative, and agree with Samuel in regarding their presence as a necessary postulate in accounting for the origin of the various forms of neurotic atrophy. This view is presented, with especial force, by Charcot.[2] The wholly negative position taken by a few authors is hardly to be justified, in consideration of the existing facts of histology and physiology. Apart from the experimental conclusions of Schiff, Meissner, Samuel, Mantegazza, and others, the region of the trigeminus is certainly proved by histological examination to contain nerves which are directly joined to cells, and in all probability govern the processes of nutrition in them. We need only point to Pflueger's investigations upon the terminations of the nerves in the cells of the salivary glands, and those of Lipmann and Klein upon the nerves of the cornea, and of the membrana nictitans of the frog. We can certainly, as Charcot has done, distinguish the secretory from the trophic nerves (in the narrower sense), and thus considerably restrict the territory of the latter.[3]

Implication of the Trigeminus.

It has been already stated that Stilling thought it necessary to assume the existence of a partial affection of the trigeminus, especially of the vascular nerves contained in the second division, in the case of Schuchardt. Still more instructive is the case observed by Axmann and Hueter, in which spastic contraction of the muscles of chewing, and a hyperæsthesia, or sharpening of the sense of temperature, preceded, upon the affected side, the development of the disease.

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[1] Arch. de Physiol. 1869. Vol. II. p. 539.

[2] Arch. de Physiol. 1869. Vol. II. p. 157 et seq.

[3] Cf. *Eulenburg*. Ueber vasomotorische und trophische Neurosen. Berlin. klin. Wochenschrift. 1872. No. 2.

The atrophy remained limited, in this case, to the parts of the face (regions of the temple and lower jaw) which are supplied by the second division, but within these limits it was extreme. The region of the left cheek and temple seemed flattened; the muscles of chewing much reduced in size; the temporal fossa almost double its proper depth; the layer of fat gone; the left half of the lower jaw much shortened and thinned; the hair on the temple and the whiskers were wanting on the left side, while they were quite abundant on the right. The last grinder tooth of the left lower jaw was wanting; the tongue was put out towards the left, and its left side was only half as broad and thick as the right. The right cheek was 6‴ thick, the left only 3‴.

In this case it is certainly natural to assume a connection between the atrophy and an affection of the trigeminus, especially its third division. The muscular branches of this division seem to have been subjected to irritation, at first frequent, then by degrees more seldom, which probably extended in like manner to the adjacent vaso-motor-trophic nerve-tubes, and involved the sensitive fibres in part. We would here mention that cases are by no means rare in which, one branch of the trigeminus (by preference the first) being affected with symptoms of sensory irritation, as a supra-orbital neuralgia, the skin and hair of the same region are also found affected. The transition from such cases, as observed by Romberg, Anstie,[1] myself,[2] and others, to certain cases of circumscribed atrophy, is quite gradual, and in describing neuralgic symptoms in conjunction with trophic, it often depends only upon the temporary preponderance of one or the other group of symptoms, or the subjective estimation of the author, whether a case shall be described as supra-orbital neuralgia with trophic disturbance, or as circumscribed atrophy with neuralgic symptoms.

The following case presents an example of this; it is given by Rômberg under the name of "trophoneurosis." A girl, aged twenty-one, having received an injury in the parietal region, became subject to attacks of pain in the left parietal region, to which was added a depression of the left temple, gradually increasing in depth, and falling out of the hair of the same region. Examination showed a furrow or depression six lines broad, beginning about one inch from the median line, and extending from the edge of the orbit straight up to the left lambdoidal suture. In the portion of this depression which passed over the parietal bone all the hair was wanting, and the eyebrow close to the foramen supraorbitale was also quite thin. Attacks of pain at regular intervals in the posterior portion of the furrow, accompanied by obscuration of the senses and a great feeling of anxiety, were present at the time of this examination.

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[1] *Reynolds*, System of Medicine. Vol. II. London, 1868. Art. Neuralgia.

[2] Lehrbuch der functionellen Nervenkrankheiten. p. 99.

The view of Baerwinkel is also noteworthy. He is inclined to place the seat of disease in the ganglia of the trigeminus. In one case, in which the region of the infra-orbital nerve was alone affected by the atrophy, Baerwinkel thinks it necessary to infer that the spheno-palatine ganglion formed the point of origin; in a second, in which the atrophy extended to all the branches of the trigeminus, the ganglion Gasseri. Since some (though not decisive) experiments seem to show that the trophic fibres which join the peripheral nerve-trunks arise wholly or in part from the spinal ganglia and that of the trigeminus, it appears that Baerwinkel's explanation of the atrophy is satisfactory; only, in the second case, it is rather surprising to see the absence of neuro-paralytic ophthalmia, which has often been observed in cases of morbid alteration of the Gasserian ganglion (Landmann, Serres, Bock, Friedreich and others).

Implication of the Facial Nerve.

It is not inadmissible to make the seventh pair of nerves assist in the explanation, since its trunk, as shown by the experiments of Schiff, Samuel, and others, probably contains trophic and vaso-motor fibres. But the assumption of Moore is decidedly erroneous, which supposes that in this affection there is a special form of progressive muscular atrophy, limited to the muscles innervated by the facialis. It is enough to point to the fact that in some cases muscular atrophy is entirely wanting; that it is at any rate a very subordinate symptom as compared with the disturbances of nutrition in the integuments, and that when present it affects the motor region of the fifth nerve (the muscles of chewing) far more than that of the facialis. Moore himself says that in his case the energy of the facial muscles seemed not at all lessened, but is of the opinion that this is owing to the fact that these muscles are not required to make any great exertion, and, therefore, a diminution of their powers cannot be easily detected.

Implication of the Cervical Sympathetic.

Until lately there seemed to be no cases of this extremely rare disease which furnished ground for tracing a relation between it and any affection of the sympathetic; attention was rather called, by the facts above stated, to the vaso-motor-trophic fibres which run with the trigeminus. But since these fibres, before passing into the trigeminus, run partly in the cervical sympathetic, the possibility remained that the latter might be involved, which found an indirect support in the circumstance that partial, slight atrophy of one side of the face has been observed in a few cases after traumatic or other lesions of the cervical sympathetic (Seeligmueller, Nicati). These were cases of *paralysis* of the cervical sympathetic. Seeligmueller describes two observations bearing on this point. In the first there was an affection of the sympathetic, in a child, connected with traumatic paralysis of the right brachial plexus from fracture of the clavicle and neck of the scapula *inter partum*. In this case there was myosis and perceptible atrophy of the right side of the face. In the second there was a gunshot wound of the left sympathetic and a part of the brachial plexus; here also there was a decided emaciation and flattening of the left cheek, in addition to the usual oculo-pupillary symptoms. Nicati regards the emaciation of the corresponding side of the face, conjoined with pallor, depression of temperature, suppression of perspiration, as distinct symptoms of paralysis of the sympathetic; he assigns them to an advanced stage or "second period." Brunner, on the contrary, considers himself bound to assume a *permanent state of irritation of the corresponding cervical sympathetic* in the very marked case of unilateral facial atrophy which he describes. The symptoms of that case corresponded entirely to the usual results of experimental irritation of the cut sympathetic of the neck or the ganglion cervicale supremum: dilatation and deficient reaction of the pupil, widening of the palpebral fissure, exophthalmus, deficiency of the secretion of tears, mucus, and sweat, lowering of the temperature of the whole left side of the face. The left ganglion supremum was also painful when pressed upon. Brunner therefore assumes that a continued irritation of the sympathetic, perhaps caused by inflammation or a tumor, produced a continued spasm of the blood-vessels, and by this means a gradual atrophy of the left side of the face. The palpitations and the epileptic fits, which were present in this case, might be traced to abnormal innervation by the sympathetic. Brunner thinks he can exclude an involvement of the trigeminus and facialis; the slight pains in the atrophic side of the face were, perhaps, explicable by muscular sensation or disturbance of nutrition.

Disregarding a few questionable points in this explanation, Brunner's case stands as yet too much alone to allow us to draw from it general conclusions in respect to pathogenesis.

Theory of Lande.

According to Lande the disease is not a neurosis, but a genuine primary *atrophy of the fatty tissue.* He considers that the fatty tissue disappears altogether, while of the connective tissue proper only the cells and fibrils perish, the elastic tissue remaining unaltered. The persistence of the latter leads again to a retraction of all the tissues, by which the skin is pressed firmly against the subjacent parts, producing the irritation of the sensitive nerve-fibres and the pallor of the skin. He seeks to explain the symptoms of cutaneous anæmia and the neuralgic or paralgic sensations—as itching—by this retraction of the elastic fibres in the absence of other connective-tissue elements. The apparent atrophy of the muscles depends, according to him, not upon a disappearance of the muscular substance proper, but only of the connective tissue which surrounds and penetrates it. The atrophy of the cartilages of the eyelids and alæ nasi, and even that of the bones, is interpreted by him as due to the changes in other tissues, especially in the blood-vessels. As the blood-vessels of the skin, in consequence of the loss of connective tissue, are compressed and lose part of their bulk, so also do the blood-vessels in the perichondrium and periosteum. These membranes, moreover, become contracted in consequence of the destruction of all their elements except the elastic tissue, and thereby not only diminish the arterial supply of the cartilages and bones, but also, by compression, aid the atrophy of these organs, which is further directly assisted by the loss of the connective-tissue elements that enter into their composition.

Not only the pallor of the integuments, but also the dryness of the skin, its rough or cicatricial feel, and the alterations in the nutrition of the hair, are explained by Lande as due to the compression experienced by the hair-follicles and the follicles of the sebaceous glands connected with them. The secretion of sweat remains intact for the most part, because the sweat-glands, lying deeper, escape compression longer.

Lande's theory is based on the presumption that the loss of cellular tissue precedes the first visible symptoms—pallor of skin, discoloration of hair, and abnormal pigmentation of the affected region, since the latter symptoms are brought about by retraction of the persistent elastic tissue and the consequent constriction of the capillaries. Lande, therefore, believes that he can entirely exclude a neurotic origin of the disease, and, regarding it as a genuine affection of the cellular tissue (tissu lamineux), he proposes for it the designation, "aplasie lamineuse progressive," or "atrophie du tissu connectif."

This assumption of Lande's has only the value of a hypothesis, for the anatomical proof of the primary and exclusive disappearance of the cellular tissue in the skin and the subcutaneous structures, bone, cartilage, muscle, etc., is by no means established. From the clinical point of view, without doubt, many objections can be raised against it. The repeated instances of occurrence after a local traumatic injury, the presence of precursory symptoms of a sensory or motor character in the region of the trigeminus, or of such forerunners as epilepsy and hemiplegia, the confinement to single nerve-districts, and, above all, the very fact of the atrophy being unilateral and sharply defined in the median line, are decidedly in favor of the neurotic origin in the majority of cases.

Diagnosis and Prognosis.

Congenital defect of symmetry in the two halves of the face need not be confounded with this disease. Although it may be very marked, so that one side of the face is decidedly smaller than the other, yet the other changes in nutrition are wanting, the color is normal, the growth of the hair unaltered. But, besides these congenital irregularities, acquired want of symmetry may be developed secondarily to deviations of the vertebral column, in torticollis or scoliosis. Especially in the so-called habitual scoliosis, consisting of a curve in the dorsal region, with the convexity towards the right, and sometimes a compensating curve of the cervical vertebræ in the opposite direction, the right side of the face is very often found the smaller. This is not the place to discuss the doubtful question of the mode of origin of this asymmetry, which is usually referred to the compression of the vessels and nerve-roots in the concavity of the cervical curve. But confusion with hemiatrophia of the face may be avoided by observing the want of change in the tint of the face, in the growth of the hair, etc., not to mention the existence of deviation.

Mistakes in diagnosis may perhaps be made in cases of arrested development of one side of the face, due to a traumatic injury received during youth. Panas[1] has described such a case, occurring in a man of twenty-five, who had suffered a fracture of the lower jaw on the left side, when ten years old, the result of which was an arrest in the development, not only of this half of the jaw, but also of the malar and superior maxillary bones. The entire side of the face therefore seemed flattened, and the nose pushed towards that side. But the color and consistence of the parts were normal in this case, and the growth of the hair quite unaltered. Such cases can hardly be taken for true atrophy of one side of the face.

A little attention will enable one to avoid confounding a hypertrophy of the opposite side of the face with this disease. In the initial stage, however, certain cutaneous affections (vitiligo and porrigo decalvans) might be thought of. In vitiligo we find the same white decoloration of the skin, the cicatricial feel, the turning gray and falling out of the hair, but not the loss of volume, which is the special characteristic of this disease. In porrigo decalvans inflammatory symptoms and œdema of the skin come first; the disease first appears in regular circular spots; the hairs fall out without previous loss of color, and finally the disease is contagious, and fungi can be demonstrated (microsporon Audouini).

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[1] Soc. de chirurgie, session of May 5, 1869.

The prognosis is very unfavorable in respect that a natural arrest is not to be expected until at least a very great loss of substance has occurred, either circumscribed or diffuse. This deformity is of the more importance, as the disease affects young people exclusively, and chiefly females. But the general health is not at all endangered by the disease.

In only one case, given by Baerwinkel, is a spontaneous improvement said to have occurred, and the face to have regained some of its fullness, on the authority of the mother of the patient, who was a girl of eight.

Treatment.

The treatment has not been successful. The internal administration of a great variety of remedies, the external use of baths, stimulating frictions, and so forth, have proved quite useless, as might have been expected. It is necessary to be cautious about frictions, as excoriations are easily produced on the atrophied parts. Almost all cases have been treated electrically for a longer or shorter time—the older ones with the rotation apparatus then in vogue, those of recent times with the volta-electric induction apparatus or the constant current. The use of the latter is said in some cases to have brought about an improvement in volume and tint, so that the power to blush returned, etc. In the case observed by Guttmann and myself, the local application of faradic and galvanic currents for several months, and the galvanization of the sympathetic, gave rise to no permanent benefit, though the local galvanization produced a reddening of the affected side of the face, which continued for several hours after each session. Brunner observed in his case that when stabile currents were applied to the two upper sympathetic ganglia, the movements of the heart instantly became slower, and the pupils dilated slightly; the affected side of the face turned red and was covered with an abundant secretion of perspiration.

BASEDOW'S DISEASE

(*Morbus Basedowii ; Graves' Disease.*)

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By the name of Basedow's disease we designate a group of symptoms, of which the chief are *palpitation with accelerated pulse, swelling of the thyroid gland*, and *exophthalmus*. A number of other disturbances, especially in the nervous system, the circulation, and (in females) in the genital sphere, are frequent symptoms, but not quite pathognomonic; sometimes they are even secondary in character. The three cardinal symptoms first enumerated are found associated in the very great majority of cases, but each one may be absent now and then, or be present in a degree scarcely passing the limits of physical health; or exophthalmus may be present alone, in which case (unlike the affection which is due to local intra-bulbar causes) it always attacks both eyes, and is combined with other general disturbances of health.

History.

Parry, in 1825, certainly described some cases of this disease under the designation of "enlargement of the thyreoid gland in connection with enlargement or palpitation of the heart," but among the eight cases mentioned by him exophthalmus is only once mentioned. St. Yves, Louis, and Demours seem to have known the disease. Basedow was certainly the first to give an accurate description, while the English usually assign the credit of the discovery to Graves. Contributions to the natural history of the disease have been made by Stokes, Charcot, Trousseau, Fischer, Genouville, von Graefe, and others. The theory of the disease has gradually assumed new forms; the first observers (Basedow, Helfft, Lubarsch, Cooper) and many since them have sought to find its essential cause in a morbid crasis, like that of chlorosis, while others (as Stokes) considered the heart the point of origin, and later observers, for the most part, have regarded the disease as a neurosis (Handfield Jones, Fletcher, Laycock), referring it especially to the cervical sympathetic (Koeben, von Graefe, Aran, Trousseau, and others), or to the spinal centres of the cervical sympathetic, the cervical medulla spinalis, and the medulla oblongata (Geigel, Benedikt). The few data of pathological anatomy have not yet given a decided result. I have explained at length in former works the relation of the several symptoms to the nervous system, and must continue to uphold the neurotic theory as a necessary postulate, while I regard the question of localization in definite sections of the nervous system (especially in the cervical sympathetic or its centres) as not yet solved, and at present unsolvable.

Synonyms.—While Basedow himself described the disease as "Glotzaugenkrankheit" (referring to the projection of the eyeballs), it has been pretty generally called by his name in Germany, as in England by that of Graves. Its two chief symptoms have given it the name of struma exophthalmica (goître exophthalmique, exophthalmic goître); the exophthalmus by itself has given the name of cachexia exophthalmica ("exophthalmos cachectique" of Fischer). Lebert proposed the name of "tachycardia strumosa exophthalmica," which at least includes the whole trio of symptoms.

Etiology.

Of the predisposing causes the influence of *sex* and *age* is first to be considered. The female sex is affected decidedly oftener than the male, in about the ratio of two to one. The middle period of life, between puberty and the climacteric years, is also affected with especial frequency. In childhood the disease is very rare, but some cases are reported; thus Devol observed it in a girl of two and a half years, Rosenberg in one of seven years, Solbrig in a boy of eight, and Trousseau in one of fourteen. It is rare beyond the climacteric period; Stokes relates the case of a woman of sixty. It is, however, necessary to add that the disease itself or its complications very often shorten life considerably.

Hereditary influence is in some cases probably of importance; but the observations on this point are not yet sufficient. In the above-mentioned case of Solbrig's, of a boy of eight years, the mother is said to have suffered from Basedow's disease.

The favorable or directly causal influence of a bad crasis of the blood, especially in *chlorosis* and *anæmia*, has been affirmed and denied with about equal frequency. To prove the dependence upon chlorosis, various authors have urged the frequency of the disease in the female sex after puberty, the frequent disturbances of menstruation during or (more especially) before the beginning of the disease, and the occasionally favorable effect of pregnancy (Charcot, Trousseau, Corlieu). But the asserted dependence upon chlorosis is contradicted by the fact of its occurrence in men, in women beyond the climacteric period, and in children, and in fresh-complexioned persons without a symptom of chlorosis or anæmia; it also sometimes develops itself acutely from accidental causes. Upon the whole, therefore, it seems better to consider the anæmia of Basedow's disease not as a primary factor, but as only a consecutive symptom, common to many chronic diseases.

The influence of a *neurotic predisposition* seems undeniable in certain cases, at least. In favor of this we have the frequency of the occurrence of the disease in connection with hysteria, to which Brueck called attention, and with epilepsy and mental disease. In a case described by Gildemeester, epileptic fits preceded the attack by several years, disappearing one year after the commencement of Basedow's disease, and not reappearing. Interesting cases of Basedow's disease, with mental disturbance, have been published by Geigel, Solbrig, Andrews, and others. I have observed several such, and once, in a woman, morbus Basedowii simultaneously with tic convulsif, and with alternate attacks of mania and melancholia.

Finally, in some well accredited cases, quite *accidental* injuries, traumatic or otherwise, seem to have caused the outbreak of the disease. Thus, mental excitement (Solbrig), violent fright (Laycock), forced cohabitation (von Graefe), injury to the head and application of leeches (von Graefe), and injury of the occiput (J. Begbie), have been stated to be causes of the sudden appearance of the disease. The importance of these causes can, however, hardly be judged of in the cases given.

Possibly climate has some influence. Lebert states that the disease is more common in North Germany than it is in Switzerland and France. I find it not common in Berlin; but it seems more so on the Baltic coast. Judging from the literature, it would seem to be thought specially frequent in England. There are, unfortunately, no statistics to appeal to.

Symptoms and Course.

The disease usually develops very slowly; yet there are exceptional cases, in which it begins brusquely and all the symptoms appear in the course of a few days. Such cases seem to be chiefly caused by accidental injuries of the above-mentioned sort (psychical excitement, or wounds). In this class belong the cases of von Graefe, Laycock, Solbrig, and others. Peter describes a case observed by Trousseau; the patient was a woman, and her disease developed itself in a single night, as a result of profound grief for the death of her father. During that whole night her nose bled profusely.

A few cases of sudden origin may also run an entirely acute course, and may recover in a short time. It may therefore be proper to separate such cases under the designation of *acute*, from the usual or *chronic* form of the disease. In the case of Solbrig the recovery was complete in ten days.

Very often the first symptoms of Basedow's disease are preceded for a considerable while by symptoms of various sorts, especially that multiform group known as hysteria; in some cases by epilepsy (Gildemeester). The first symptom is usually *palpitation*, which at first occurs only at intervals, but by degrees becomes more permanent, and, as it were, habitual. It is conjoined with *acceleration of the pulse*, which at first may not be constant, but afterwards may become permanent, without remission, so that the pulse maintains a continuous, uniform, quite enormous rate. I observed for a long time a patient who had a pulse of 144 every day and at every hour of the day, with scarcely any variation at all. In lighter cases the rate averages from 90 to 120 beats; in others it may reach 200 (M'Donnell[1]), and in one observed by Gildemeester it was at times too rapid to count.

It is a characteristic fact that the ordinary remedies for lowering the pulse often produce no effect. In a few cases the palpitations and the changes in the rate of the pulse are entirely absent. In fifty-eight older ones collected by von Dusch, this was the case three times. More lately Chisolm, Leube, and others have contributed observations bearing upon this point. In Leube's case the rate reached 82 only once; in Chisolm's it was 85, while there were no palpitations at all.

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[1] Dublin Journ. of Med. Science. Vol. XXVII. p. 203.

Physical examination of the heart reveals nothing abnormal, as a rule. The most that is found is an occasional systolic murmur, usually plainest at the apex, and probably only indicative of anæmia. Other circulatory phenomena have been observed: these are occasional epigastric pulsation, increased force of the pulse in the carotids and their larger branches, especially the thyroids, and a whirring and blowing that may be heard by auscultation before the struma is developed, pulsation of the retina (Becker), and in rare cases pulsation of the liver. Lebert, who observed this phenomenon three times, regards the hepatic pulse as arterial, and distinguishable from the venous by its slighter intensity; it is the less likely to be confounded with the ordinary epigastric pulsation, as it is oftenest felt just over the right lobe of the liver.

When these symptoms of disordered circulation have lasted for some weeks or months, the second chief symptom, *struma*, is developed, slowly or rapidly, often within the course of a few days; its usual form is that of a swelling of soft, elastic consistency, distributed uniformly over the whole thyroid gland. It is less common to find only one lateral lobe affected; in other cases both are involved, but one more so than the other. Very rarely the struma appears before the palpitation; still more rarely it is wholly absent—according to von Dusch, in three cases out of fifty-eight. The surface of the tumor is often marked by veins, greatly dilated and crowded with blood; the hand laid upon it feels almost everywhere a distinct frémissement, and auscultation reveals loud blowing sounds, often increased during systole. These symptoms may subsequently diminish or disappear, while the tumor assumes a somewhat firmer and more resistant consistency. This seems to depend on the fact that the early hyperæmia and dilatation of vessels yield at a later period to an actual hyperplasia of the glandular tissue. The struma of Basedow's disease seldom reaches a considerable extent. It is also worthy of note that the size of the tumor is subject to frequent changes;

mental disturbance, the action of medicine, various changes in the other conditions of the disease, and above all, pregnancy, have an unmistakable influence. According to Stokes, Graves observed two cases, in which each attack of palpitation was accompanied by a swelling of the thyroid gland, which disappeared when the attack was past. Similar cases, in which the tumor was observed to increase or decrease according to the force of the beat of the heart, have been reported by Henoch, Begbie, Marsh, Gildemeester, and others.

Exophthalmus makes its appearance soon after the struma—in a few cases before it—and still more rarely as the initial symptom, preceding the struma and the palpitations (in one case of Mackenzie's and one of Chvostek's). It is almost without exception bilateral. A few cases of the unilateral sort are mentioned (Mackenzie, Foerster quoted by Lebert, Schnitzler, Chisolm, Emmert), but some suspicion must remain in respect to the diagnosis of these cases. (See "Diagnosis.") Sometimes the exophthalmus comes earlier in one eye than in the other, and often it is not equally developed upon both sides. Sometimes it may be wholly wanting (in four out of fifty-eight cases, according to von Dusch), while in other instances it forms the only cardinal symptom (Praël, Dégranges, and Sichel, quoted by Fischer), in which cases it is associated with other sorts of general disturbance, so that the terms "exophthalmie cachectique" or "cachexie exophthalmique" seem not unsuitable.

In degree, the exophthalmus varies extremely; sometimes there is but a slight prominence of the eyeball, sometimes the protrusion is so excessive that no part of the globe is covered by the eyelids; and even luxation of the globe may occur (Pain quoted by Trousseau). The prominent eyeball has an unusual lustre and a peculiar stiffness; in fact, if the deformity has existed long, it often loses its mobility in great part or wholly. The exophthalmus is not always equally distinct, but often increases and disappears, proportionately to the force of the pulsations of the heart, and sometimes may be diminished by light pressure on the eyeball. It is almost always accompanied by a phenomenon, first observed as characteristic by von Graefe: *the upper lid loses its power to move in harmony with the eye-*

ball in the act of looking up or down. This want of agreement between the movement of the lid and the raising or depressing of the point of view is not caused by the exophthalmus as such, for the movement of the lid is not impaired in exophthalmus occurring from other causes, such as tumors of the orbit, while in Basedow's disease, if ever so little developed, it is weakened or suspended. That this phenomenon is independent of the exophthalmus is further shown by the fact that it may disappear during the course of the disease, without any improvement having taken place in the latter, and this may be a spontaneous result or may follow the use of narcotic injections. According to von Graefe, the phenomenon is therefore pathognomonic, and is extremely important in diagnosticating slight degrees of the disease, in cases where the eyes hardly project more than the normal amount, and where there is no struma. He observed a patient whose only symptoms were this defective agreement between the movements of the lid and the raising or lowering of the glance, and palpitation of the heart; the diagnosis of Basedow's disease was made upon this evidence alone. I have seen no such cases; but in one case, with great protrusion of the globe, I have seen the movement of the lid almost normal, scarcely diminished beyond physiological limits; in like manner also, in another case, with moderate exophthalmus. I, therefore, do not incline to grant a pathognomonic value to this sign, although its very great frequency must certainly be admitted.

Another and a rarer group of symptoms must perhaps be regarded in some cases as secondary, as a sequence of the exophthalmus and the defective power to depress the upper lid. These symptoms are *disturbances in the nutrition of the bulb*, especially in the outer coats of the eye, the conjunctiva and cornea. The impairment of the power of moving the lid prevents the eye from receiving its due share of moisture, whence come dryness in the conjunctival sac, distention of the conjunctival veins, and conjunctivitis. The lachrymal secretion is often increased. In the majority of cases nothing worse occurs; but in bad cases severe disturbance of nutrition of the cornea follows, closely resembling what occurs in neuroparalytic ophthalmia, and perhaps explicable in the same way. (See below, "Analysis of the

Symptoms.") The cornea first loses its sensibility; on its surface dry yellow spots appear, which gradually enlarge, and the final result is the formation of an eschar followed by diffuse desiccation or even perforation. In other cases infiltrations occur at several points of the cornea at once, and ulceration or even perforation occurs. It is remarkable that these disturbances in the nutrition of the globe are common in men (Basedow, Praël, Naumann, von Graefe), although in a few instances also observed in women (Lawrence, Tatum, Teissier).

The *accommodation* is usually unaffected in Basedow's disease, though sometimes weakened in consequence of deficient mobility of the globe. *Ophthalmoscopic examination* usually shows, according to von Graefe, a dilatation and increased tortuosity of the retinal veins. Becker observed, besides this, *spontaneous pulsation of the retina* in two cases. In the immense majority of cases the *pupil* is quite unaltered—a fact of the more importance, as the exophthalmus produced experimentally in animals, as in death by suffocation or by paralysis of the heart, is always accompanied by dilatation of the pupils. It is true that the statements of authors regarding the pupil are not quite in accord; some authors (Romberg, Reith, Geigel, Friedreich, Cazalis quoted by Trousseau, Fournier and Ollivier) speak of dilatation of pupils, but this was probably not connected with Basedow's disease, as the patients in question were probably myopic. Gildemeester in one case found the pupils at first much dilated, afterwards contracted; Cheadle found a slight dilatation with entirely regular reaction; Nitzelnadel in one case contraction; Emmert in some cases contraction, in others dilatation. Von Graefe, however, informed the author that among nearly two hundred cases of Basedow's disease he had not once found dilatation of the pupil. Neither have I myself seen it in cases of genuine morbus Basedowii; but I have had occasion to state that there are cases of struma with slight unilateral exophthalmus and accelerated pulse, which cannot be taken as cases of Basedow's disease, but as *secondary neuroses of the cervical sympathetic, caused by the strumous tumor*, characterized by considerable dilatation of the pupil, and by the elevation of the temperature on the affected side of the head (see "Diagnosis").

Among the less constant symptoms of Basedow's disease must be mentioned the *elevation of the bodily temperature*, which did not escape the attention of the first observers. The patients feel this, even when it is very slight, as a sense of heat, often accompanied by an *increased secretion of sweat*. Basedow himself pointed out this feeling of heat, and in later literature cases are described in which it was so severe that the patients threw off their clothes (Trousseau, Fournier and Ollivier). As a rule, the elevation observed by instruments is not very considerable; but it appears rarely wanting when the test is applied carefully and repeatedly. Paul found in one case an elevation of from 0.5° to 1° C.; Teissier often from 1° to 2°; Cheadle, in his report of eight cases, says that he always found elevation of temperature. In all the cases of my own observing I have found an elevation of from 0.5° to 1° C., at least temporarily; and in one case, that of a girl of twenty years, whose temperature was taken very often for the period of three-quarters of a year, it always stood from 38.2° to 38.8° (100.8° to 101.8° F.) in the axilla. But Charcot and Dumont report cases in which the temperature was entirely normal. This elevation is of course only important in the uncomplicated cases, which are not accompanied by febrile disease.

There is also a series of *nervous disturbances*, not to be considered as essential and pathognomonic, but as quite frequent accompaniments, and probably based in part upon an existing neuropathic predisposition. Among these must be included the complications with hysteria, epilepsy, and insanity, already enumerated under the head of etiology; it must also be said that the slighter degrees of mental alteration, a changeable and especially a lachrymose disposition, alternations of excitement and depression, etc., are of extreme frequency in the higher grades of Basedow's disease, especially in women. The patients often complain of severe headache, sometimes limited to one side; a feeling of dizziness, complete inability to work, weakness of thought and memory, and tormenting sleeplessness, and express a fear of losing their minds. Sometimes there is excessive appetite; oftener loss of appetite, even aversion to all food, nausea, and vomiting, and, in consequence, emaciation. All these symptoms, and especially the mental ones, are not usually present in an equal degree, but may increase, diminish, or at times disappear, proportionally to the leading symptoms of the disease.

The frequent occurrence of disturbed menstruation, particularly amenorrhœa, has been mentioned already. In a case recently related by Leube a slight cutaneous sclerema of the face and the backs of the hands was observed. Nitzelnadel and Chvostek[1] give cases in which a sweating upon one side (ephidrosis unilateralis) was observed to coincide with contraction of the corresponding pupil; and a case of hyperidrosis unilateralis, with struma and hypertrophy of the left ventricle, recently observed by Ebstein,[2] may be classified here.

The *duration* of the disease, if we except those rare and in many respects anomalous cases classed as acute Basedow's disease, is always very protracted. Months and years may pass, with alternate improvement and relapse. Not very rarely an entire recovery occurs, spontaneously or in consequence of therapeutic measures. In some few cases pregnancy seems to have had a favorable influence (Charcot, Trousseau, Corlieu). But usually the progress is toward the worse. Consecutive changes in the heart often develop; the permanently increased labor exacted of the heart leads to dilatation of both ventricles and compensating hypertrophy, and a disturbance in the compensation may, at a later time, hasten the fatal issue. In other cases death occurs during exhausting marasmus or intercurrent diseases (as œdema of the lungs or apoplectic attacks), or other complications, as especially tuberculosis of the lungs and valvular difficulty of the heart. In the case described by Fournier and Ollivier, and in another by Rabejac (cf. "Anatomical Changes"), death occurred during progressive gangrene of the lower extremities, without any apparent cause for this sudden suspension of nutrition.

Anatomical Changes.

The number of reports of autopsies is very small, which need not surprise us, as patients with Basedow's disease seldom enter hospitals, and still more rarely remain there till their death. The anatomical examination of the struma has usually shown a dilatation of the veins, or considerable development of the arteries (Smith and MacDowell, quoted by Stokes, Naumann, J. Banks, Moore, Fournier and Ollivier); the glandular tissue is either normal, or in a state of simple hyperplasia, or filled with cysts and altered by new formations of decidedly a secondary character.

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[1] Wiener med. Wochenschrift. 1872. Nos. 19 and 20.

[2] *B. Fraenkel*, Diss. Breslau, 1874.

Behind the globe of the eye a considerable development of fat is almost always found (Basedow, Heusinger, Naumann, Laqueur, Traube and Recklinghausen, Peter, Fournier and Ollivier, and others); sometimes with this there is atheromatous change of the ophthalmic artery (Naumann), or, as in Traube's case, advanced fatty degeneration of the eye-muscles, probably caused by disuse and stretching. More attention has lately been paid to the nervous system, and especially, since the well-known experiments of Claude Bernard, to the sympathetic in the neck and its ganglia. Upon this point the reports vary greatly. While most authors have obtained positive results (Trousseau quoted by Peter, Reith, Cruise and McDonnell quoted by Moore, Traube and Recklinghausen, Biermer, Virchow, Geigel, Knight), others have not been able to show any changes in the sympathetic and its ganglia (Paul, Fournier and Ollivier, Rabejac, Wilks).

The case described by Peter, remarkable for the suddenness of its commencement (see above), ended fatally in eight days. At the autopsy (by Lancereaux), the chief morbid appearance was a predominance of the connective tissue, and a diminution of the nerve-elements in some parts of the cervical sympathetic, more in the right and less in the left inferior ganglion, while the upper and middle ganglia seemed quite normal. The cardiac plexus presented no striking alterations.

The case of Reith was a man of twenty-four; the disease had lasted a long time. The principal result of the autopsy (Beveridge) was to show an enlargement of the middle and lower cervical ganglia; they were hard and firm, and under the microscope showed an infiltration with a grayish mass. The cord of the sympathetic, also, and the branches sent off from it to the lower thyroid and the vertebral artery, were enlarged and in a state of tuberculous change.

In the case examined by Cruise and McDonnell, the lower cervical ganglia of both sides were almost obliterated, and replaced by cellular and fatty tissue. In the case of Traube and Recklinghausen a striking thinness of the sympathetic and its ganglia is spoken of; no other changes were to be found in the sympathetic or vagus. Biermer mentions in a letter the case of a man in whom there was great atrophy of both sympathetics, especially the right. Virchow found in a man who died with dropsical symptoms, a hypertrophy of the heart, with very extensive myocarditis and enlargement of the thyroid gland, and also a very considerable enlargement and interstitial thickening of the cervical sympathetic, especially in the upper and lower ganglia.

In Geigel's case (a man of forty-eight) both cervical sympathetics were found ensheathed in remarkably thick connective tissue, abounding in fat, but the microscope discovered no change in the nerves proper, nor, except intense brown pigmentation, any in the ganglia; neither was there to be seen any increase of interstitial connective tissue. The cervical parts of both vagi, both in the fresh condition and after preservation in Mueller's fluid, were normal. The following changes were also found: obliteration of the central canal of the spinal cord, much fullness of the small and of the minute vessels of the cord; in the direct neighborhood of the central canal the medullary substance was quite hard, and there was a slight proliferation of the neuroglia; there was also a tumor on the synchondrosis spheno-occipitalis.

In Knight's case (a man of thirty-three), the left lower cervical ganglion of the sympathetic was larger than the right, the amount of connective tissue quite large, while the nerve-cells were decidedly smaller and less pigmented than usual. In the middle and upper ganglia of the sympathetic of the left side the nerve-cells were much smaller than on the right. The nerve-fibres in the left sympathetic were one-half the size of those of the right.

Paul found nothing abnormal in the thoracic and cervical portions of both sympathetic cords; nothing in the lower cervical ganglion. In the fresh condition, as well as after staining with carmine, the nerve-fibres and ganglion-cells appeared of entirely normal dimensions, with distinct nuclei and nucleoli, some colorless, some pigmented.

In the oft-quoted case of Fournier and Ollivier there were found no changes in the cervical, thoracic, and abdominal cord of the sympathetic. The examination was made with great care by Ranvier upon carmine preparations. Both the nerve-fibres and the ganglion-cells were entirely normal; increase of connective tissue was nowhere present. In Rabejac's case (a woman of fifty-eight) the sympathetic was examined microscopically by Bouvier, and found entirely normal. Wilks found no coarse changes in the ganglia of the sympathetic, which even seemed unusually white; microscopically there was found nothing abnormal except an increase of the connective-tissue fibres.

These few facts, so heterogeneous in character, must not be applied to the pathogenesis and theory of the disease without the greatest care. It is true that there are only four negative to eight positive reports regarding the sympathetic; but some of the latter are such as hardly involve necessary disturbances of the functions of the sympathetic. Thus, in Geigel's case there was only a thickening of the connective-tissue sheath, while the nerve-fibres, ganglion-cells (except for the pigmentation), and interstitial connective tissue were normal. It seems to me worthy of note that in the few carefully examined cases where there were positive changes in the sympathetic, the lower ganglion is spoken of with considerable unanimity as the one chiefly or exclusively affected (Peter, Reith, Cruise and McDonnell, Virchow, Knight). With the lower ganglion the middle one has once been found affected (Reith), and the upper one once (Virchow); in Knight's case the upper and the middle ones were altered, but less so than the lower. It must be remarked that Ebstein, in the case of hyperidrosis unilateralis with struma and hypertrophy of the left side of the heart, already mentioned, found striking changes in the left cervical sympathetic, chiefly in the lower ganglion, consisting of varicose swelling of the blood-vessels with much pigmentation of the ganglion-cells. By the kindness of Dr. Ebstein I myself had opportunity to confirm this fact at the Breslau meeting of naturalists. In most cases of undoubted Basedow's disease the alterations occurred in both sympathetics, but in some the affection was not equal, being more developed on the right side (Peter, Biermer), or the left (Reith); in Knight's case the left sympathetic seems to have been affected exclusively. In Geigel's case there were also changes in the spinal cord, which do not admit, however, of a direct interpretation.

Analysis of the Symptoms, and Theory of the Disease.

Let us now attempt an analysis of the leading symptoms, with the object of forming a general theory of the disease; we shall be obliged, at the best, to content ourselves with a somewhat unsatisfactory result.

We will first speak of the *struma*, because the attempt has often been made to make the other symptoms depend upon it (Koeben, Piorry, Cros, and others), although the struma is by no means the first symptom in the majority of cases. Trousseau and others considered the struma (as they did the exophthalmus) to be the consequence of a congestive state of the upper half of the body, while the disturbed menstruation, or amenorrhœa, was supposed to depend on a deficient supply of blood to the lower half of the body. The cause of the hyperæmia was sought, in obedience to the analogy furnished by Bernard's experiments, in a dilatation of the vessels, owing to paralysis of the vaso-motor nerves running in the cervical sympathetic. Certainly there are some facts which support this explanation: the small arterial branches of the carotid, pulsating strongly, often tortuous and visibly prominent; the frequently rapid growth of the tumor, its softness; the perceptible pulsation of the thyroid arteries, the blowing sounds audible over them; the alternate enlargement and diminution of the tumor proportionate to the force of the pulsation of the heart, and the abnormal development of the thyroid arteries and veins, as shown by the pathological anatomy. On the other hand, there is as yet no experimental evidence that a section of the sympathetic can produce struma. This proof must be insisted upon, especially as struma is a very common complaint in animals, and even occurs endemically in the regions where men are affected with it (Virchow, Baillarger). Boddaert also has proved that tying the internal and external jugular veins and the inferior thyroids in rabbits and guinea-pigs produces a swelling of the thyroid gland. The origin of Basedow's struma may therefore be regarded as very probably due to a dilatation of the arterial and venous vessels; its connection with paralysis of the vaso-motor nerves (or with the sympathetic) must, however, be regarded for the present as matter of question.

Of late, an explanation directly opposed to this has been offered by Benedikt. He regards the dilatation of the vessels as due, not to paralysis, but on the contrary to irritation of the dilator nerves, which also run in the sympathetic. He bases this view upon the recent experiments of Bernard, Schiff, Ludwig, and Lovén, which seem to prove the existence of nerves which dilate the vessels actively. These experiments, it is true, relate only to a few nerve-districts (nervi erigentes penis, according to Lovén; the nerves of the parotid which arise from the facial; muscular arteries, according to Ludwig and Hafiz), and mostly admit of another interpretation. Neither can we satisfactorily explain the mechanism of such an active dilatation of arteries consistently with their histological structure. Benedikt attempts such an explanation by pointing to the longitudinal muscle-cells in the wall of the vessels, the shortening of which over the tense column of blood he thinks must cause a shortening of the arterial tube, with dilatation. Whether this view agrees with the fact will perhaps be shown by later observations; in the large arterial vessels the active dilatation cannot certainly occur in the manner described, as they possess no muscular cells. As regards the thyroid arteries, which are specially concerned in the production of struma, Moeller, at the suggestion of Benedikt, has examined them, and has found between the intima and the adventitia, as well as in the latter itself, a pretty thick layer of smooth muscular cells. Certainly Benedikt's explanation, if correct, would render the theory of the disease extremely simple (see below).

The second cardinal symptom, the *exophthalmus*, is probably referable to various genetic factors. Doubtless it is largely dependent upon *venous hyperæmia and increased development of fat in the cellular tissue of the orbit.* The existence of abnormal hyperæmia during life is rendered probable by the analogous condition existing in struma, and also by the observations which show that the exophthalmus often diminishes when the palpitations of the heart grow less, and, vice versa, increases when the palpitations are worse; while the globe of the eye is further observed to be easily pushed back by the finger, and after death to recede into the orbit spontaneously.

In favor of the possibility of a mechanical forcing forward of the eyeball, we have many analogies. In new-born children a slight exophthalmus is observed after a tedious labor, due to pressure and checked circulation; the same is observed after instrumental labors; also in the mother, when she has made violent efforts in parturition. Exophthalmus may also arise from various other causes,[1] quite mechanical in their nature, which lead to serous infiltration of the retro-bulbar fatty tissue (as nephritic dropsy), and also from congestion of the head.[2] A variety of other causes, not including tumors of the orbit—such as violent exertion, convulsions, etc., may cause exophthalmus from increased blood-pressure in the orbital veins (Demarquay).[3] With these cases may be classed a remarkable observation of Decès,[4] who saw an exophthalmus of the left eye appear in a woman after violent congestion of the head, and disappear again when the neighboring temporal artery grew larger, and the covering skin became red and puffy. The results of ophthalmoscopic examination in Basedow's disease also justify the assumption of venous hyperæmia. And lastly, Boddaert has quite recently produced a considerable exophthalmus in rabbits and guinea-pigs, by artificial intra-ocular hyperæmia due to tying of the two internal and external jugular veins, with section of both cervical sympathetics. This lasted for some days, and did not disappear until the venous hyperæmia had become reduced, owing to the establishment of collateral circulation.

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[1] *Demarquay*, Traité des tumeurs de l'orbite, p. 171 and 183 (cases of Roché and Lecorché).—*Fischer*, l. c. (observations XVII. and XVIII.).

[2] *Demarquay*, l. c. p. 189 (observation of Sichel).

[3] L. c. p. 157 et seq.

[4] Thèse sur l'anévrysme cirsoïde; cf. Gaz. hebdomadaire, 1862, p. 482.

The second cause of exophthalmus, the considerable increase in the quantity of fatty tissue behind the eyeball, has been directly proved by a series of autopsies, and I have convinced myself in one very marked case that this factor certainly plays a leading part in the development of the symptom. Perhaps a third cause ought to be added to these two, namely, the *contraction of the smooth muscles of the orbit*, composing the musculus orbitalis, discovered by H. Mueller, which lies in the neighborhood of the fissura orbitalis inferior, and is innervated by the sympathetic. Claude Bernard has shown that section of the cervical sympathetic, or of the anterior roots of the two upper dorsal nerves, produces retraction of the eyeball, while galvanic stimulation of the peripheral ends after section causes dilatation of the palpebral fissure and exophthalmus. This exophthalmus is probably mainly caused by contraction of Mueller's muscle. The latter is very poorly developed in man, and is opposed by very powerful antagonists, the straight muscles of the globe; but I do not think that the circumstance has been sufficiently attended to, that the latter muscles undergo a gradual fatty degeneration in Basedow's disease, as I found in the above-mentioned case; so that their antagonistic action is greatly weakened or lost. H. Mueller has, moreover, discovered other smooth muscles in the upper and lower lids, which may assist in protruding the bulb by enlarging the palpebral fissure. That these muscles receive their nerves from the sympathetic is proved by R. Wagner's and Mueller's experiments upon executed criminals, in which electrical stimulation of the cervical sympathetic caused the eyelids to open. Remak had previously shown (in 1855) that irritation of the cervical sympathetic in animals produces a raising of the upper lid. Finally, Sappey[1] has described smooth muscles in the orbital aponeurosis, which are supposed to assist in protruding the bulb when the cervical sympathetic is irritated.

The theory which explains Basedow's exophthalmus by the action of the above muscles encounters a considerable difficulty, owing to the fact that a permanent tetanic contraction of these muscles must be assumed, a state for which we have no certain physiological analogies. It may also be properly doubted whether the force of this weak muscle is adequate to produce an exophthalmus of so marked a character as often exists in Basedow's disease. Even the most powerful experimental irritation of the cut cervical sympathetic, in animals, is not able to bring about so decided a protrusion. But, on the other hand, it must be remarked that the degree of this protrusion varies a good deal from time to time, and these variations may perhaps be referred to corresponding oscillations in the condition of tonic stimulation of the sympathetic fibres which supply the muscle. Another circumstance has much weight in confirming this theory, namely, the fact that von Graefe's symptom is usually associated with the exophthalmus; that is, *a want of agreement between the movement of the lid, and the raising or depressing of the glance.* The cause of this insufficiency in the motion of the lid is said by von Graefe to be due to anomalous innervation (spasmodic contraction) of the smooth muscles of the orbit, which receive their innervation from the sympathetic, since these parts probably regulate the movement of the lids which is associated with the shifting of the plane of vision.

A close examination of these questions would lead too far. I am compelled to believe, in view of all these facts, that the venous congestion and retro-bulbar growth of fat certainly play the chief part in Basedow's disease, but that the participation of the smooth orbital muscles innervated by the sympathetic cannot be wholly excluded. Another question, not yet answered, is, whence the venous congestion and the increase of orbital fatty tissue come; and whether these may not also be connected with the vaso-motor trophic fibres running in the sympathetic. (See below.)

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[1] Académie des sciences, session of October 21, 1867.—*Prévost* and *Jolyet*, ibid. Nov. 18, 1867 (Arch. gén., Jan. 1868, p. 104).

The third cardinal symptom of the disease (in point of time usually the first), the *palpitation with acceleration of the pulse*, was once commonly supposed to be a symptom of the anæmia or chlorosis. I need only refer back to what has already been said in disproof of this idea, as it has at the present day but few supporters. Stokes's view, also, which makes the palpitations symptoms of a primary heart-disease, needs no further refutation. Of late the increased activity of the heart in Basedow's disease has been considered due to a disturbance of the functions of the cervical sympathetic. In the trunk of the latter, as von Bezold has shown, run the excito-motor fibres, which originate in the central nervous system—the fibres which accelerate the action of the heart. Irritation of the cervical sympathetic increases the rate of the pulse; the assumption of a permanent condition of irritation in the cervical sympathetic would, therefore, explain satisfactorily the increase in the contractions of the heart. Such an assumption of course encounters the difficulty of supposing that there can be a permanent condition of irritation in certain groups of nerve-fibres. Friedreich has attempted a somewhat different explanation. He thinks that the vaso-motor nerves which originate from the sympathetic are in a state of paralysis, which may produce a dilatation of the coronary arteries, an increased flow of blood to the muscle of the heart, and thereby an increased excitement of the ganglia of the heart. According to this view the accelerated action is caused directly by increased activity of the automatic ganglia of the heart, and only indirectly by the sympathetic. But this view does not differ essentially from the former, for the sympathetic nerves of the heart are commonly supposed to have the function of transferring the excitement coming from their centre to the ganglia of the heart. Whether this transference is caused by an increased flow of blood to the ganglia, or by an increased irritation of the sympathetic fibres which are connected with them, is certainly a subordinate point in relation to the question before us.

A theory of the disease, to deserve the name, ought to be able to derive all the symptoms, or at least the three cardinal ones, from a common source. We have already seen that this demand is not satisfied by the hypothesis of a primary chlorotic basis (Basedow), or a primary cardiac affection (Stokes). A better success ought to be expected of the neurotic theories, especially that which assumes a primary implication of the cervical sympathetic or its spinal centres. Some have sought to derive all three symptoms from an affection of the sympathetic; some, only the exophthalmus and the palpitations, leaving the struma as an independent affection, or even calling it the cause of the sympathetic lesion. The last hypothesis with which we shall have to do, originates with Koeben. He considers that the struma exercises pressure upon the cervical sympathetic, which causes exophthalmus. Piorry and Cros entertain the same view. But it is contradicted by the fact that the struma often appears at the same time with the exophthalmus, and sometimes later, even many years later (Desmarres[1]), or may be wholly wanting in spite of the existence of exophthalmus in a high degree. Besides, the ordinary struma, which occurs endemically and is often harder and bulkier than that of Basedow, produces no exophthalmus, or but a very trifling amount. In cases where compression of the sympathetic by tumors is proved to exist (struma or enlargement of lymphatic glands), the exophthalmus is wholly wanting, or is quite trifling in amount (see below, "Diagnosis"), while in such cases considerable mydriasis exists, which is not observed in Basedow's disease. Finally, the exophthalmus does not diminish in proportion to the diminution of the struma, as would be expected if it were due to the effect of pressure. In one case Charcot found the exophthalmus equally intense, although the tumor had disappeared and the rate of the pulse had become normal. And, *vice versa*, Taylor saw the exophthalmus disappear and the general health improve, without any diminution in the size of the tumor.

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[1] *Fischer*, l. c. observation III.

The referring of all symptoms to a primary affection of the sympathetic has been attempted especially by Aran and Trousseau, upon the basis of Bernard's experiments. Against this very tempting view there is one chief difficulty: certain symptoms (especially the struma) are such as are commonly supposed to follow experimental section, or paralysis of the sympathetic; others, on the contrary, might be caused by galvanization, and may be presumed to indicate a permanent condition of irritation in the sympathetic. This difficulty would certainly be removed if Benedikt's assumption could be established, which refers the struma to an active irritation of dilator nerves which run in the sympathetic. But this assumption, as we have seen, lacks at present an adequate support. There remains, moreover, a second objection, already stated, which lies in the difficulty of conceiving of a permanent state of irritation continued for years together.

While Benedikt seeks to regard the struma as a symptom of irritation, and thus to bring it into an analogy with the other leading symptoms, Friedreich, on the contrary, as we saw above, interprets the palpitations as a symptom of paralysis of vaso-motor nerves. The exophthalmus might likewise be referred, at least in part, to a vaso-motor paralysis which causes congestion of the orbit, and in this way an apparent agreement might be reached. Finally, it may be allowable to suppose that the cervical sympathetic may be at one and the same time in a state of irritation and of paralysis, that some fibres are irritated while others are placed in a paretic condition by the causal lesion. Affections of peripheral nerves afford analogies of apparently opposite conditions existing together in the same nerve. For example, in neuritis there may be symptoms of irritation in the motor fibres with diminished sensibility, and *vice versa*. In the same fibres we often find apparently opposed conditions: in the sensitive, a diminution and an increase of sensation (anæsthesia dolorosa); in the motor, diminution and increase of movement (paresis or paralysis with spasms and contractures).

The difficulties that lie in the way of assuming an involvement of the sympathetic have led several authors, especially Geigel and Benedikt, to place the seat of the disease farther up in the cervical medulla or the medulla oblongata. Claude Bernard has shown that the oculo-pupillary and the vaso-motor nerves which run in the sympathetic have centres, not in one part of the cord, but at different levels. Section of the anterior roots of the two first spinal nerves produces (according to Bernard) only the oculo-pupillary symptoms, for the fibres which originate in the cilio-spinal centre leave the spinal cord at this point; but section of the ascending cord of the thoracic sympathetic between the second and fourth ribs produces only the vascular and thermic effects, dilatation of the vessels and elevation of temperature upon the side operated on. Geigel assumes that the point of origin of the lesions of Basedow's disease is to be sought in these two centres, and that one of them—the oculo-pupillary—is in a condition of irritation; while the other—the vaso-motor—is in a state of paralysis. In truth, this does not solve the problem, but only transfers it from the peripheral trunk of the sympathetic to the sympathetic centres in the cervical medulla spinalis. And why, in two centres so near each other, opposite conditions should prevail for years together, is no less difficult to explain than their co-existence in different fibres of the cervical sympathetic itself.

Benedikt places the seat of the disease in the medulla oblongata, and not in the cervical sympathetic, because fibres are often affected which belong lower down in the sympathetic; for when a variety of nerves, originating from the central nervous system at various points, take part in one affection, that affection must be located in that part of the nervous system where all the fibres involved lie near together, and that part is here the medulla oblongata. Against this argument it must be said that the symptoms on which it is based (disturbances of the abdominal organs, amenorrhœa, etc.) are by no means necessarily connected with vaso-motor fibres running in the sympathetic. They are, rather, complications or secondary symptoms, dependent on the abnormal action of the heart, etc., and they constitute no urgent reason for regarding the centre of the vaso-motor nerves in the cervical cord as the original seat of the disease.

The data furnished by pathological anatomy are partly in favor of, partly against the supposition of a primary affection of the sympathetic. If we regard the question for a moment as decided in the positive sense, it cannot be denied that the remaining subordinate symptoms can be explained without violence from this point of view. The want of agreement between the movements of the lid and changes in the plane of vision has already been explained. The ophthalmia is perhaps due in part to the insufficient protection given by the lid, and the consequent want of moisture of the globe; but this can hardly account for the whole, since the same unfavorable state of things exists in paralytic lagophthalmus (*e. g.*, after facialis palsy), without, as a rule, affecting the eye at all. Basedow's ophthalmia is probably analogous to the so-called neuro-paralytic form occurring after section of the trigeminus, and the loss of sensibility of the cornea, which occurs in severe cases, especially favors this view. Since fibres of the sympathetic enter into the trunk of the trigeminus, it cannot be said to be impossible that Basedow's ophthalmia may be a result of disturbances of innervation of these fibres. Whether the fibres thus implicated are vaso-motor or trophic (as Meissner is inclined to think in respect to neuro-paralytic ophthalmia) must remain for the present unsettled, owing to the uncertainty of our present knowledge of trophic nerves. Charcot[1] regards neuro-paralytic ophthalmia, like other trophic disturbances, as a phenomenon of *irritation*, a consequence of inflammatory stimulation, like that which is developed in the nerve after partial section, as in the experiments of Meissner and Schiff.

The *lachrymation*, which is a frequent symptom, may be referred to a change of innervation of the fibres which pass from the sympathetic into the first branch of the trigeminus. Perhaps this constant wetting of the conjunctiva with tears explains the rarity of the occurrence of malignant inflammations of the cornea in Basedow's disease. The *ephidrosis unilateralis*, which is present in a few cases, might also be referred to a paralysis of the vaso-motor fibres that run in the sympathetic, especially as in the cases described (Nitzelnadel, Chvostek) myosis of the same side is said to have existed.

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[1] Leçons sur les Maladies du Système Nerveux. Paris. 1872-3.

Stellwag explains the *dilatation of the pupil*, in the few cases in which it co-exists with Basedow's exophthalmus, as due to a paralysis of the pupillary branch of the oculo-motor nerve, consequent upon cerebral neuro-paralytic dilatation of vessels. The isolated paralysis of this branch, the other fibres of the motor oculi remaining unaffected, is referred by him to the fact that the branches destined for the pupil do not join the other oculo-motor fibres until after the latter have crossed the crura cerebri, and that they have been proved to originate from several centres of various function. Yet the dilatation of the pupil, if present, may be explained much more easily by referring it to an irritation of the dilator fibres of the sympathetic, and the contraction which exists in other cases to a paralysis of the same fibres; and yet, assuming a sympathetic origin, the almost unvarying absence of dilatation of the pupils presents a great difficulty, for changes in the diameter of the pupil are the most constant symptom in other lesions of the cervical sympathetic, such as mechanical or traumatic injuries.[1] I wish to call attention again to the fact that certain autopsies show a preponderance of affection of the lower cervical ganglion, while the upper and middle ones are scarcely, if at all, affected. It is possible that this may constitute the reason why mydriatic symptoms are usually absent in Basedow's disease.

Diagnosis and Prognosis.

It is evident tnat there may be difficulty in making the diagnosis, especially at the beginning of the disease. We shall have to infer the existence of the disease in some cases, even in the absence of one of the three leading symptoms, and even where only exophthalmus is present, provided it be bilateral and accompanied by general disturbance. Among the subordinate symptoms, the feeling of heat, the elevation of temperature, the want of agreement between the motions of the lid and depression of the visual plane, possess a certain diagnostic value. It must also be remembered that, of the leading symptoms, some may not only be quite absent, but may temporarily disappear, while the others persist, and that their order of appearance may also vary very much.

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[1] See *Eulenburg* and *Guttmann*, Pathologie des Sympathicus. pp. 5 and 6.

As regards differential diagnosis, cases of *primary struma with consecutive irritation of the sympathetic* are of special importance. In such cases a moderate degree of exophthalmus and a more or less considerable amount of palpitation, with accelerated pulse, may very closely resemble Basedow's disease.

A case which clearly belongs to this class is related by H. Demme (mydriasis and slight exophthalmus in a man with cystic struma). At the autopsy the cervical cord of the sympathetic was found upon the left side reddened, and with the surrounding connective tissue in a state of serous infiltration. There was no microscopic change. I have for a long time observed a case which belongs to this category. It was that of a young lady, very slender, who had a tuberculous affection of the apices of the lungs. She had a vascular struma, almost confined to the right side, with a very much developed mydriasis, a perfectly immovable iris, and a moderate exophthalmus of the right eye, with paresis of accommodation; a permanent depression of temperature could also be demonstrated in the auditory meatus of the affected side, which was 0.3° or 0.4° C. lower than the other. Chills frequently occurred towards evening, during which the face was pale. The pulse was very quick (128–140); forcible palpitations of the heart; loud, blowing murmurs over the struma, increased during systole.

Calabar bean acted favorably for a time upon the mydriasis and the paresis of accommodation; the symptoms were also temporarily relieved during the local use of galvano-puncture, which produced for a time a considerable reduction in the size of the tumor, and also a lessening of the rate of the pulse by from twenty to thirty beats per minute.

A means of establishing a differential diagnosis between this neurosis of irritation, produced mechanically by struma, and genuine Basedow's disease, is afforded by the *unilateral symptoms of pupillary and vascular irritation*, viz., mydriasis, paresis of accommodation, depression of temperature in the ear of the affected side; in true Basedow's disease the exophthalmus is bilateral almost without exception, the pupillary symptoms are absent, the temperature of the meatus is either unchanged or shows a uniform elevation corresponding to that of the axilla.

The *prognosis* will be seen to be unfavorable for the most part. Yet the possibility of a spontaneous disappearance, or of removal by medical treatment, is not to be excluded. The prospect of succeeding in this will be the greater, the more satisfactory is the general health of the patient, and the more certainly we can exclude organic changes of the heart, dilatation, valvular lesions, etc., or a neuropathic tendency and severe disturbances of innervation.

Treatment.

The earliest attempts at treatment were based upon the belief that the disease depended on anæmia or chlorosis. Remedies were therefore chosen which are supposed to improve the composition of the blood, to give strength, and to relieve chlorosis—above all, *quinine* and *iron*. Though the belief upon which this theory was founded can hardly be defended at the present day, yet it cannot be denied that, when these remedies are used properly and perseveringly, a surprising success is occasionally attained, even if there were failure in a good many cases. Traube's method may be regarded as the best. It consisted, as he himself has told us, in combining quinine and iron in such a way that each of the two remedies is used alternately for about three weeks; the quinine in a moderate dose (five grains per diem), and the iron in the form of Vallet's pilular mass. Traube has obtained very favorable results from this method in a series of cases in which the whole of the symptoms were relieved for years at a time, the patient enjoying perfect health.

Besides these so-called strengthening remedies, those which depress the pulse (*digitalis*, *veratrin*) have been much used. Experience has shown that these drugs, which are efficient in other diseases, often fail of their action in Basedow's disease (von Graefe, Geigel, Cerf Lewy, Fritz, and others), and that their therapeutic value is really little or nothing. The debilitating modes of treatment, such as blood-letting, derivation, preparations of iodine and arsenic, often seem to produce direct harm. As regards the forms of *iodine*, which seem to be demanded for the relief of struma, it is said that they do effect some benefit in this direction, but that they also increase the palpitations, and in some cases have done a good deal of harm. Smith has recently seen good results from the use of *belladonna*, and ascribes the effect to the stimulus it exercises upon the sympathetic.

The cervical sympathetic has recently been directly treated by *galvanization with the constant current*, in accordance with the theory of a primary affection of the sympathetic. Von Dusch appears to have been the first to treat a case for a considerable time in this way, and he succeeded in reducing the rate of the pulse from 130 to 70 or 64 beats, and in diminishing the exophthalmus.

My own first experiments in this method of treating the sympathetic in Basedow's disease were made in 1867, upon a lady aged fifty—sent to me for treatment by von Graefe—who had an habitual pulse of 108–130, with abnormal tension of the carotids. When her cervical sympathetic was galvanized by placing the negative pole upon it, with a very weak current (from 6 to 8 elements), the pulse was observed to sink gradually to 84, or even to 70, while the tension of the carotids and radial arteries continually diminished. The patient felt much better at the same time. Since then I have employed galvanization of the sympathetic in six other cases of this disease, mostly very severe; but, as the patients came from a distance, they could remain but a short time under treatment, and the results, as respects both the improvement of the leading symptoms and the general health, were therefore but small. But I was able to ascertain with certainty that at each session, after a short application of electricity, the rapidity and force of the heart's action were considerably lessened, and sometimes a marked soothing influence upon the mind was observed. As a rule, my method was to apply the negative pole to the cervical ganglia of each side in turn, or to both at once by means of a divided electrode. In a few cases the struma was attacked at the same time by *galvano-puncture;* but I have seen only doubtful and transient results from this practice, though it was usually very well borne.

Chvostek has recently made some very thorough observations upon the action of galvanization on the sympathetic. Thirteen cases were treated by this method, mostly for a long time, all of which were considerably improved, or even nearly cured; and this, sometimes, after a very few applications. Galvanization had an important influence, especially in procuring the recession of the exophthalmus and the struma, while its effect in the way of lessening the action of the heart was very slight in several cases. M. Meyer reports equal success; he obtained so decided an improvement in four cases of quite marked Basedow's disease, that only a few traces of the leading symptoms remained. In addition to the manifest success of galvanization in reducing the struma and exophthalmus (the pulse remaining as high as before), Meyer speaks strongly of the benefits accruing to the general health, as shown by relief to the chlorotic symptoms and reappearance of the menses in normal quantity and character.[1] Leube also observed a considerable improvement in the above-mentioned case, during galvanization of the cervical sympathetic.

A careful regulation of the habits is of special importance in Basedow's disease: avoidance of all excitement and of great physical exertion, as well as of coitus; a mild, nutritious diet largely composed of milk and vegetables, an entire avoidance of all exciting drinks, such as coffee, tea, and alcoholic beverages; a life largely spent in the open air, especially in the country, or in mountain health-resorts of moderate elevation. Among the *mineral springs* those containing iron deserve the preference; long and repeated cures in Franzensbad, Pyrmont, Schwalbach, etc., are sometimes of evident value. But the whey cures and grape cures, though still excessively popular, do not deserve the recommendations they get, if only for the injury they often inflict upon the general health.

Of the individual symptoms, those which affect the eye may require special local treatment. For the exophthalmus, von Graefe recommended painting with tincture of iodine between the eyebrows and the upper lid, or frictions with ointment containing iodide of potassium, compresses, local electrization, and in severe cases even tarsorrhaphia as a protection against malignant affections of the cornea. If the latter appear, the eye must be protected from all injury, and covered with wet compresses; in some cases it may be well to employ tarsorrhaphia.

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[1] *Perres*, on the contrary, observed in one case which was treated by galvanism only a considerable diminution of the pulse, while the struma remained unaltered.

PROGRESSIVE MUSCULAR ATROPHY.

(Atrophia muscularis progressiva.)

Van Swieten, Commentarii in H. Boerhave aphorismos. III. 1750. p. 370.—*Abercrombie*, Pathological and practical researches on diseases of the brain and the spinal cord. Transl. into German by Gerhard von dem Busch. 1829.—*Darwall*, London Med. Gaz. VII. 1831. p. 301.—*Bell*, The nervous system of the human body. London, 1830. Transl. by Romberg under title Physiologische und praktische Untersuchungen des Nervensystems. 1832.—*Romberg*, Klinische Ergebnisse. Berlin, 1846.—*Dubois*, Gaz. méd. de Paris. 1847. No. 47. p. 926.—*Duchenne*, Session of Acad. de méd. May 21, 1849.—*Romberg*, Lehrbuch der Nervenkrankheiten. 1850. III. p. 159. 3. Aufl. 1857.—*Aran*, Recherches sur une maladie non encore décrite du système musculaire (atrophie musculaire progressive). Arch. gén. de méd. t. XXIV. Sept. and Oct. 1850.—*Thouvenet*, Thèse sur la paralysie musculaire atrophique. Dec. 1851.—*Romberg*, Klinische Wahrnehmungen und Beobachtungen. Berlin, 1851.—*Meryon*, Med. Chir. Trans. 2d ser. vol. 7. London, 1852. p. 81.—*Duchenne*, Union médicale. 1852; Bull. de thérapeutique. 1853. p. 295, 407, 438.—*Cruveilhier*, Arch. gén. Mai, 1853. p. 561; Gaz. méd. de Paris. 1853. No. 16.—*Bouvier*, Gaz. méd. de Paris, 1853. No. 15. p. 232.—*Landry*, Ibid. No. 17. p. 261.—*Burq*, Gaz. des hôp. 1853. No. 53.—*Nièpce*, Gaz. méd. 1853. No. 17. p. 260; Arch. gén. Mai, 1853. p. 626.—*Chambers*, Med. Chir. Transactions. XXXVII. 1854. p. 19.—*Guérin*, Arch. gén. Mai, 1854. p. 626.—*Cohn*, Günsburg's Zeitschr. f. klin. Med. V. 1854. p. 360.—*Schneevogt*, Niederl. Lancet. Sept. and Oct. 1854.—*Virchow*, Handbuch der speciellen Pathologie und Therapie. I. Erlangen, 1854. p. 322.—*Betz*, Prager Vierteljahrschrift. XLIII. 1854. p. 104.—*Robin*, Comptes rendus des séances de la soc. de biologie. 2e sér. I. 1854. p. 5.—*Oppenheimer*, Ueber progressive fettige Muskelentartung. Diss. Heidelberg, 1855.—*Hasse*, Krankheiten des Nervensystems. Erlangen, 1855.—*Wachsmuth*, Henle und Pfeufer's Ztschr. f. rat. Med. VII. 1855. p. 88.—*Gros*, Gaz. des hôp. 1855. No. 50.—*Eisenmann*, Canstatt's Jahresbericht, 1855. III. p. 86.—*Valentiner*, Prager Vierteljahrschrift. XLVI. 1855. p. 1.—*Virchow*, Arch. f. path. Anat. 1855. VIII. p. 537.—*M. Meyer*, Wiener

med. Wochenschr. 1855. Nos. 41 and 42.—*Diemer*, Günsburg's Zeitschrift. 1855. VII. 1.—*Cruveilhier*, Arch gén. de méd. Jan. 1856. p. 1.—*Eulenberg* (sen.), Deutsche Klinik. 1856. Nos. 11–14.—*Reade*, Dublin Jour. of Med. Science. Nov. 1856. p. 399.—*Vigla*, Gaz. des hôp. 1856. No. 146.—*Helfft*, Allg. med. Centralzeitung. 1856. No. 12.—*Moussons*, Gaz. des hôp. 1857. No. 108.—*Barton*, Dublin Hosp. Gaz. 1857. June 15.—*Frommann*, Deutsche Klinik. 1857. No. 33. p. 317.—*Friedberg*, Pathologie und Therapie der Muskellähmung. Weimar, 1858.—*Roberts*, An essay on wasting palsy. London, 1858.—*Baerwinkel*, Prager Vierteljahrschrift LIX. 1858. p. 133.—*Sandahl*, Hygiea. 1858.—*Rodet*, Union méd. 1859. No. 26.—*Luys*, Gaz. méd. de Paris. 1860. No. 22.—*Leubuscher*, Krankheiten des Nervensystems. 1860.—*Legendre*, Gaz. méd. de Paris. 1860. No. 23. p. 365.—*Duchenne*, Électrisation localisée. 2e éd. Paris, 1861.—*Dumenil*, Gaz. hebd. 18 Janv. 1861. p. 38.—*Anstie*, Med. Times and Gaz. Febr. 1861.—*Clarke* and *Gairdner*, Beale's Arch. of Med. Oct. 1861. p. 1. vol. III.—*Gull*, Guy's Hosp. Rep. 3d series. VIII. p. 244. 1862.—*Remak*, Oesterr. Zeitschr. f. prakt. Heilk. 1862. p. 1 and 29.—*M. Meyer*, Deutsche Klinik. 1862. No. 7.—*Hemptenmacher*, De aetiologia atrophiae muscularis progressivae. Diss. Berlin, 1862.—*Guthzeit*, De novissimis observationibus ad atrophiae muscularis progressivae naturam et therapiam spectantibus. Diss. Berlin, 1862.—*Malmsten*, Hygiea. 1862. p. 555.—*Lockhart Clarke*, Brit. and For. Med. Chir. Rev. July, 1862. p. 215; Beale's Archives of Med. vol. IV. 1863. p. 26.—*Vulpian*, Union méd. 1863. No. 49. p. 159.—*Friedreich*, Ueber degenerative Atrophie der spinalen Hinterstränge. Virchow's Archiv. 1863. XXVII. p. 1.—*Voisin*, Gaz. hebd. 1863. No. 37; Gaz. des hôp. 1863. No. 110. p. 437.—*L. Meyer*, Virchow's Archiv. 1863. XXVII. p. 419.—*Trousseau*, Gaz. des hôp. 1863. Nos. 12 and 14.—*Foerster*, Handbuch der speciellen path. Anatomie. 2. Aufl. 1863.—*Ruehle*, Greifswald. med. Beiträge. II. 2.—*Duchenne*, Comptes rendus. 18. Janv. 1864. p. 168.—*Jaccoud*, Bull. de la soc. méd. des hôp. 1864; Union méd. 1865. t. XXV. No. 4. p. 60.—*Remak*, Application du courant constant au traitement des névroses. Paris, 1865.—*Schueppel*, Ueber Hydromyelus. Archiv der Heilkunde. VI. 1865. p. 295.—*Bergmann*, Petersb. med. Zeitschrift. VII. 1865. p. 83.—*Brugnoli*, Gaz. med. Lombard. 1866. No. 23.—*Duménil*, Gaz. hebdom. 1866. Nos. 4–6.—*Fieber*, Die diplegischen Contractionen. Berl. klin. Wochenschrift. 1866. No. 25. p. 261.—*Menjaud*, Gaz. des hôp. 1866. No. 3. p. 10.—*J. Simon*, Nouveau dictionnaire de méd. (Art. "Atrophie musculaire progressive.") Paris, 1866. t. IV. p. 27.—*Baudrimont*, Journal de Bordeaux. Mars, 1866. p. 148.—*Clarke*, Med. Chir. Transactions. 1866. XLIX. p. 171; 1867. L. p. 489.—*Duménil*, Gaz. hebdom. 1867. No. 29.—*Swarzenski*, Die progressive Muskelatrophie. Berlin, 1867.—*Trousseau*, Clinique méd. de l'hôtel Dieu. 2e éd. German transl. by Culmann. 1868.—*L. Clarke*, Med. Chir. Trans. LI. 1868. p. 249.—*M. Meyer*, Die Elektricität in ihrer Anwendung auf praktische Medicin. 3. Aufl. Berlin, 1868.—*A. Eulenburg*, Ueber diplegische Contractionen gelähmter Muskeln. Centralblatt f. d. med. Wiss. 1868. No. 3.—*Eulenburg* and *Guttmann*, Pathologie des Sympathicus. Archiv f. Psychiatrik und

Nervenkrankh. I. 1868.—*Landois* and *Mosler*, Neuropathologische Studien. Berl. klin. Wochenschrift. 1868.—*Benedikt*, Elektrotherapie. Vienna, 1869.—*J. Grimm*, Virchow's Archiv. XLVIII. 1869. p. 445.—*v. Bamberger*, Wiener med. Presse. 1869. Nos. 27 and 28.—*Nesemann*, Berliner klinische Wochenschrift. 1868. No. 37; 1869. No. 52.—*Ollivier*, Des atrophies musculaires, thèse. Paris, 1869.—*Erb*, Deutsches Archiv für klin. Medicin. V. 1869. p. 82.—*Charcot* and *Joffroy*, Arch. de la phys. normale et pathologique. II. 1869. p. 354.—*Hayem*, ibid. p. 263.—*de Silva Lima*, Union. méd. 1869. No. 141.—*Stein*, Deutsches Archiv f. klin. Med. VI. 1869. p. 593.—*Leyden*, Archiv f. Psychiatrik und Nervenkrankheiten. II. 1870. p. 648.—*A. Eulenburg*, Virchow's Archiv. XLIX. 1870. p. 446.—*Joffroy*, Gaz. méd. de Paris. 1870. No. 10. p. 129.—*M. Rosenthal*, Handbuch der Diagnostik und Therapie der Nervenkrankheiten. Erlangen, 1870.—*Wilks*, Guy's Hosp. Rep. 3d series. XV. 1870. p. 1. *M. Rosenthal*, Allg. med. Centralzeitung. 1871. No. 7.—*Gerhardt*, Berlin. klin. Wochenschrift. 1871. No. 23. p. 265.—*A. Eulenburg*, Virchow's Archiv. LIII. 1871. p. 361.—*Hallopean*, Arch. de méd. Sept. 1871. p. 277, 305.—*Hammond*, Treatise on Diseases of the Nervous System. New York, 1871.—*Kussmaul*, Ueber die fortschreitende Bulbärparalyse und ihr Verhältniss zur progressiven Muskelatrophie. Sammlung Klinischer Vorträge. No. 54. Leipzig, 1873.—*Friedreich*, Ueber progressive Muskelatrophie u. s. w. Berlin, 1873.—*Charcot* (Leçons cliniques sur les maladies du système nerveux), Klinische Vorträge über Krankheiten des Nervensystems. German trans. by Fetzer. Stuttgart, 1874.—*Leyden*, Klinik der Rückenmarkskrankheiten. I. Berlin, 1874.—*Markusy*, Zur Lehre von der progressiven Muskelatrophie und progressiven Bulbärparalyse. Diss. Breslau, 1874.

The chief of this group of symptoms is *a wasting of the voluntary muscles, which is gradual, progressive, extends to a more or less considerable part of all the apparatus of motion, and usually observes a very distinct order of march.* This definition excludes at once all those forms of muscular atrophy which originate in constitutional diseases (as that occurring after such acute diseases as typhoid fever, and that in constitutional diseases and cachexias like cancer and syphilis). It also excludes those which can be shown to be dependent upon loss of motor innervation, or which depend on congenital defects, arrests of development or growth, and which usually affect simultaneously all the tissues of one extremity, one-half of the body, etc. Progressive muscular atrophy is further a *severe disease, ending with the complete annihilation of the functions of the muscles affected, very rarely curable, and in many cases leading directly to* *death.* It is characterized anatomically by the nature of the pathological changes in the muscles, consisting in *chronic myositis, interstitial proliferation of connective tissue with secondary destruction of the muscular fibres, and finally, fibrous degeneration;* the anatomical changes in the nervous system, however, do not at present admit of so simple and comprehensive a statement of their nature and significance.

History.

Swieten, in describing lead-palsy (1754), gives an account of a case of this nature, without explaining it. In the first half of our century such cases were repeatedly given by Abercrombie, Darwall, Charles Bell, Romberg, Graves, Dubois, and Duchenne, mostly without any careful distinction of their character.

The great Charles Bell[1] has made some interesting remarks in connection with a case named by him a local palsy of the muscles of the extremities, but which must certainly be counted in with the disease we are describing. He says:

"These affections of particular muscles, or classes of muscles, imply a very partial disorder of the nerves. A disease of the brain, or a disease in the course of the nerve, must influence the whole limb, or that portion of it to which the nerve or nerves are distributed. But in these cases particular subdivisions of the nerves included in the same sheaths, or running the same course, are affected. I am inclined to attribute such partial defects to the influence of visceral irritation. In that case it must still be the influence of the sympathetic nerve which produces it; and yet, on the other hand, it seems impossible to account for such entire loss of motion without the intermediate influence of the brain."

The hint contained in this argument received, at first, little attention. Even Romberg, who described three cases of the sort in his Lehrbuch (1850), reckons them among the "paralyses dependent on the spinal cord as a conductive apparatus," and treats of them accordingly under the title of spinal palsies. Abercrombie also makes only the very general remark that this must be an obscure local nervous affection; Darwall says the same. Duchenne first called attention to the anatomical changes in the muscles, discovered by himself, and in a memoir addressed to the Paris Academy in 1849 characterized them as "atrophie musculaire avec transformation graisseuse."

In spite of predecessors like these, Aran, in 1850, spoke of the disease (and not without justification) as a "hitherto undescribed" affection of the muscular system, for which he proposed the term "atrophie musculaire progressive." He sought to place the essential cause of it in an excessive irritability of the muscular system, which led to its fatty degeneration, without previous involvement of the nerves, saying: "Le travail morbide est primitivement et uniquement dans le système musculaire." Quite otherwise did Cruveilhier conceive of the disease, claiming for himself a priority of description, and naming it "paralysie musculaire atrophique," or "paralysie musculaire progressive atrophique." He regards the paralysis as due to the muscular atrophy, but explained the latter as dependent upon atrophy of the anterior roots of the spinal nerves, or, indirectly, upon degeneration of the gray central substance of the cord, from which he said that the anterior roots originated. The proof of this opinion was presented chiefly in the autopsy of the rope-dancer Lecomte: "dont le nom restera attaché à l'histoire de cette maladie." (See "Anatomical Changes.")

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[1] Phys. und path. Untersuchungen des Nervensystems. Deutsch von Romberg. 1832. p. 364. ["The Nervous System of the Human Body." London, 1830, case LXXXVIII., p. clxii.—Trans.]

Since Aran and Cruveilhier first led the way with their works, the generality of authors who have treated of progressive muscular atrophy have been divided into two great parties. Some, with Aran, regarded the disease as a primary muscular affection—a *myopathy*, although in detail they differ more or less widely from Aran's views. Of such are Meryon, Oppenheimer, Hasse, Friedberg, and Duchenne, the latter of whom, as we have seen, pointed out the fatty degeneration of the muscles before Aran, and proposed the designation "atrophie musculaire graisseuse progressive." Others, like Cruveilhier, laid stress on the *neurotic* character of the atrophy, its dependence on and relation to certain parts of the nervous system. In this regard, it is true, the widest differences of opinion have arisen; single autopsies, or purely theoretic deductions, have served as the basis for assuming as the primary seat of disease, in one case the peripheral nerves (Guérin); in others, after Cruveilhier, the anterior roots (Bouvier, Gros, Valentiner, and others); or again, the spinal cord (Eisenmann, Cohn, Leubuscher, Baerwinkel, Joffroy, Ollivier, Hayem, Gull, Lockhart Clarke, Grimm, et al.); or finally, the sympathetic with its ganglion (Schneevogt, Remak, Jaccoud, J. Simon). We cannot follow out in detail the discussions which have been had upon this point, and which were sustained with more vigor formerly than now. Very recently, Friedreich has fully and thoroughly established the myopathic theory, which he had previously defended. The weight of his arguments and observations is undeniable; and yet there may be many weak points which he has failed to cover, and it is certainly quite premature to regard the neuropathic theory as defeated. The latter has now resolved itself, with most of its defenders, into the assumption of a *primary disease of the motor nerve-cells, or ganglion-cells of the anterior cornua*, which are supposed to exercise a trophic influence on the voluntary muscles. This theory, supported by the results of autopsies made by Luys, Duménil, Clarke, Schueppel, Hayem, Charcot and Joffroy, and others, numbers some important adherents, including Hammond; Leyden[1] also seems to recognize it, and of late even Duchenne appears to lean towards it. It finds an eloquent and ingenious supporter in Charcot, who refers progressive muscular atrophy to a chronic irritative atrophy of the anterior ganglion-cells, which are gradually, successively, and progressively attacked. (Cf. "Theory of the Disease.") Although I cannot deny that the neuropathic theory, and especially Charcot's form of it, seems to me the most satisfactory, and the one best in accordance with the pathological facts and the physiological assumptions; and although in another place[1] I have stated it as my own belief, nevertheless, I hope in the following pages to speak without prepossession, and to do equal justice to the different points of view, especially as I regard the question as entirely unripe for a present decision.

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[1] Klinik der Rückenmarkskrankheiten. I. pp. 123, 154.

Several synonyms remain to be mentioned in addition to those named above. These are: Paralysie amyotrophique; myopathic paralysis, or paralysis ex alienata musculorum nutritione (Friedberg); paralysis atrophica (Eisenmann); paralysie rhumatismale périphérique du mouvement (Guérin). All these names are unsuitable, as they imply a "paralysis"; they have not found acceptance.

Etiology.

Various authors (Meryon, Roberts, Trousseau, and others) have believed in the existence of a *diathesis, a predisposing anomaly of constitution*, as forming the basis of progressive muscular atrophy, but they have not been able to state exactly what constituted this diathesis. Friedreich defined it more distinctly as "*a nutritive and formative weakness of the muscular tissue, causing an increased tendency to irritative and degenerative disturbances of nutrition in the said tissue.*" This definition is based upon the myopathic theory, which has been so remarkably presented by Friedreich. Other authors, in accordance with the neurotic theory, believe that they can explain the disease by reference to a neuropathic predisposition, such as must be assumed in many cases of hemicrania, of angina pectoris, etc., and consider that this view finds a powerful support in the frequent combination with other unquestionable neuroses, as progressive bulbar paralysis, tabes dorsalis, mental disease, especially dementia paralytica. They look for this predisposition in an originally defective formation of certain regions of the central nervous system, probably those masses of ganglion-cells in the cord which are regarded as centres of muscular nutrition. It were needless to dispute this view in detail; at present nobody seems able to give a concrete statement of his notions about it, and we must confine ourselves to rather vague descriptions. It is far more important to make clear the point that a diathesis of some sort or other must be assumed to exist in at least a large number of cases. Among the facts which support this view, that of *heredity* is of the first importance. Roberts, in 1858, was able to prove a hereditary tendency in eighteen out of sixty-nine cases, and Friedreich mentions four from his own observation. Especially interesting is a case described by Hemptenmacher, in which the disease could be traced far and near among the ramifications of a connection, composed of three families, which were united by repeated marriages and could all be traced back to one pair of ancestors living a hundred and fifty years ago. In this case only males were attacked, and the women were spared, even when they were themselves the medium of transmission of the disease. In another case, of which Friedreich[1] also traces out the family relationships with exactness, the disease was inherited not only by male, but also by female members of the family; a woman transmitted it to her children, though they were the product of three distinct marriages. Trousseau mentions a family in which the great-grandfather, grandfather, father, and son, suffered from the disease, the course of which closely coincided in all the generations. A circumstance which is sometimes connected with heredity is the appearance of *several cases in one family*, especially among children of one father and mother; yet the same occurs in some cases where inheritance cannot be proved, or where at least both parents and other ancestors were healthy. Thus, Meryon describes a case in which four brothers were attacked by the disease; Eulenburg senior described the history of twin brothers who, without any visible cause, were attacked simultaneously and in exactly the same manner in their eighteenth year by progressive atrophy of the lower extremities.

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[1] Ueber vasomotorische und trophische Neurosen. Berl. klin. Wochenschrift. 1872. No. 2.

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[1] Ueber progressive Muskelatrophie, etc. p. 43. Obs. XII.

In a case which has but lately come to my notice, affecting the family of a Count L——, out of seven children, two brothers and two sisters were attacked, while the remaining three brothers were spared. The brothers were aged from eighteen to twenty years when attacked; one was an officer, the other a law student; of the sisters one was older, the other younger. The parents were perfectly healthy, but an uncle had suffered from epileptic dementia. In another place I have related the case of three sisters, separated in age by intervals of three or four years, in whom the disease developed itself at exactly the same period (the eighth year) and took the same course, beginning with the lower extremities.[1] In this case no hereditary tendency could be proved.

As regards the influences of *sex* and *age*, the male sex shows a decidedly greater tendency than the female. In the latest and most comprehensive statistics, those of Friedreich, in 176 cases only 33 were females, that is, about 19 per cent.; of 28 cases of which I possess notes, 17 were in men, 11 in women. It would be natural to ascribe this to the greater amount of severe and exhausting labor performed by men, which, in fact, appears to constitute one of the causes in a certain number of cases; but this explanation does not suit in cases where the disease appears in childhood, and attacks only the male children of a family, whether there be a provable hereditary taint or not. Here I will merely point to the case above mentioned, described by Hemptenmacher from Friedreich's Clinic, in which all the female members of the family remained unaffected. The same was the case also in Meryon's observations: in one, four sons were attacked, and seven daughters remained unaffected; in another, eight boys were attacked, while the four sisters were not. In the case described by Eulenburg senior, that of the twin brothers, their sisters were not attacked. It might therefore be supposed that the original defect in the formation of the muscular system, or of certain parts of the central nervous system, is produced or transmitted oftener in the male than in the female sex. Yet in some instances the opposite fact seems to exist, the female children in one family being exclusively affected.[1]

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[1] Virchow's Archives, vol. LIII., Heft 2 und 3, p. 361.

As regards the influence of age, the period of middle life (from 30 to 50 years) is usually spoken of as the most frequently affected. Statistics certainly agree with this view. Wachsmuth, among 49 cases, found 13 under the age of 5 [? 15], 8 from 15 to 30, 22 from 30 to 50, and only 6 over 50 years. Ehrhardt's and Friedreich's figures are similar. Of my own patients, as far as could be ascertained, 7 acquired the disease before the age of 10, 6 before their 20th year, 2 before the 30th, 8 before the 40th, 5 before the 50th, and no one later. It follows that a considerable percentage falls within the period of youth, about one-half within the first thirty years, and almost the same number in the years of middle life, while the development of the disease in advanced age seems quite exceptional. Upon examining the cases singly, it appears that those dependent on a hereditary basis, or upon any congenital pre-formation, come to view at an earlier period of life, usually before the close of the 20th year, while individuals who have no specific diathesis, and whose disease owes its origin to the accidental injuries, hereafter to be described, for the most part belong to the middle period of life, which is the most exposed to these influences.

Among the other causes which favor or perhaps directly provoke the origin of the disease, *acute exhausting diseases*, and certain constitutional *dyscrasiæ*, such as *lead intoxication* and *syphilis*, must be mentioned. Not a few instances stand recorded in which the disease is said to have developed during the convalescence from acute disease, as after typhoid (Moussons, Benedikt, Gerhardt), after measles (Eulenburg senior, Nesemann), after acute articular rheumatism (Anstie, Friedreich), cholera with very protracted typhoid (Friedberg). In the case of the twin brothers, the disease followed an attack of measles which affected both at once. Charcot and Joffroy have seen the disease occur immediately after childbed. Other authors have laid it to excesses in venery, or onanism (Aran, Oppenheimer,

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[1] While writing these words, three sisters were brought to me from another family, suffering from the disease, while their brothers were well.

Diemer). In Johnson's and Clarke's case the disease is said to have developed in a child in consequence of vaccination. Probably these and similar circumstances would not have had any influence but for the existence of a predisposition. As regards lead-poisoning, a diffused affection of the muscles may certainly be one of the symptoms of chronic saturnism, and this affection may bear a great resemblance to a certain rare group of cases of progressive muscular atrophy, namely, the multiple or generalized form. Yet it were advisable, if only upon grounds of general fitness, completely to separate this saturnine affection of the muscles from progressive muscular atrophy in the narrower sense. The same may be said of those rarer cases of diffused disease of the muscular apparatus which are said to have originated in the action of constitutional lues, and which improve or get quite well under the employment of antisyphilitic remedies, as iodide of potassium (Nièpce, Rodet).

Among the more direct causes, exhausting muscular activity is to be mentioned, in the first place, however few in number may be the cases in which excessive and unwonted efforts can be proved to have been a cause. In favor of this view is the fact that progressive muscular atrophy seizes upon certain muscles and certain groups with a distinct predilection, and in an almost regular order of succession, preferring the upper extremities, and usually beginning with the right, and of the right selecting certain muscles of the hand and the ball of the thumb; moreover, the fact of the great preponderance of the male sex and the laboring class. There are also some cases in which the outbreak of the disease has been preceded by a great strain upon the power of the muscles first or principally affected. Betz observed atrophy of the right side three times in smiths and saddlers, who had to do heavy work with their right hands; Gull observed the same in a tailor after excessive exertion. In one, mentioned by Hammond,[1] the disease is said to have been caused by an excessive use of one thumb and finger in playing faro! In some of those rarer cases, in which the left upper extremity was the first attacked, there were special circumstances; thus, in Friedreich's case of a dragoon, who may have exhausted his left hand by holding the bridle in riding; in a worker in morocco-leather, who used to press hard with his left hand (Voisin); in a musician, who for four years played several hours a day on the bass-viol (Schueppel). And in the cases in which the disease did not begin in the hand, but in the muscles of the shoulder or the lower extremities, an excessive use of the muscles attacked could sometimes be shown; thus Schneevogt mentions two cases of primary atrophy of the shoulder muscles, especially the deltoid, one of the right side, in a sailor who had to pump for days together while the ship was leaking; the other of the left side, in a woman who always carried her child on her left arm while suckling it. Friedreich mentions a case in which continued threshing was considered to be the cause of atrophy of the right arm and shoulder, and the scapular and thoracic muscles of the same side. Leinweber tells of a musketeer who, in consequence of handling his piece, was attacked by atrophy of the left pectoralis and serratus magnus.

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[1] Psychological and Medico-legal Journal, Sept. 1874, p. 175.

A primary affection of the lower limbs, commonly beginning with the lumbar muscles, is most frequent in children (as Roberts stated), and seems to be due to the preponderant use of these muscles in standing and walking, in playing in a sitting and bent posture. In the cases of diffused disease (Friedreich's "general muscular atrophy"), the influence of universal exhaustion, of excessive use of a great number of muscles, may sometimes be traced, as in a case by Vigla. In all, Roberts was able to trace the effect of hard labor as a factor in twenty-five cases out of sixty-nine. As to how this acts we are totally in the dark. From the myopathic point of view we can imagine that an excessive increase of functional physiological stimulation in a muscle may become a direct cause of disease, as is the case in other organs, and that perhaps certain products of decomposition, occurring in excess during exhausting muscular activity (carbonic acid, lactic acid), exercise an irritating chemical action upon the muscular tissue. Yet the exhausting muscular toil is not sufficient by itself to explain the disease in certain persons, but has to be helped out by supposing a congenital or acquired disposition—a favoring individual diathesis.

Besides exhausting labor of the muscles, *rheumatic* and *traumatic* lesions have been particularly blamed as direct causes. It is plain that these two circumstances will often coincide, for persons who have to work hard with their hands are particularly exposed to atmospheric and traumatic influences. Yet some cases seem to show the directly favorable influence of exposure to cold and wet. Thus, Duménil saw atrophy of the lower extremities appear after long-continued standing in the water in fishing for trout; Friedreich, in the case of a man who was frequently exposed to the cold while chopping wood in the forest in winter. E. H. Richter saw a total atrophy of the hands in a man who had suffered from severe sweating of the hands, and used therefore to bathe them in ice-cold water and snow; Menjaud saw this after long-continued work in the wet and cold. It is of course right to separate from these the cases, more frequent in number, in which severe exposure to cold has caused a partial paralysis with secondary wasting. As regards traumatic influences, they may be either lesions of the nerve-trunks with secondary atrophy of the muscles, or direct lesions of the muscular substance (*e. g.*, by crushing). The latter forms are designated by Friedreich as *primary traumatic muscular atrophy*, and he relates a very instructive case in which, after the hand had been crushed, the atrophy extended progressively upwards over the entire upper extremity, and finally led to the complication of bulbar paralysis.[1] I have formerly described a case in which the hand was compressed for several hours, after which symptoms entirely similar to muscular atrophy appeared.[2] Finally, there occur cases in which inflammatory irritation seems to be propagated from neighboring organs (especially the shoulder and hip joints), or cicatrices or suppurating wounds appear to be the cause—cases which are grouped together by Friedreich as "myopathia propagata." Friedreich relates several cases of this sort[3]; but they are hardly to be considered as cases of progressive muscular atrophy in the narrow sense, and deserve mention here only as showing the possibility of the origination of extensive myopathies, *per contiguitatem*, from peripheral foci of irritation and inflammation, without any previous lesion of the corresponding nerve-trunks and without the intervention of neuritis descendens. Whether it be proper to include here the cases cited by Clarke, Hasse, Friedberg, and others, in which a person who has fallen from a railway carriage, or been thrown by his horse, etc., becomes a victim to atrophy of certain groups, especially the muscles of the shoulder, must remain an open question, since the injury in such cases may equally well have fallen upon the peripheral nerves.[1]

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[1] L. c. p. 236. Observ. XXIV.

[2] Zur Galvanopathologie und Therapie der Lähmungen. Berlin. klin. Wochenschrift. 1868. No. 2.

[3] L. c. p. 152. Obs. XVIII. and XIX.

Symptoms and Course.

The cardinal symptom, though often by no means the only one, is the *atrophy* with its gradual development and its successive extension to certain groups of muscles; the diminution in bulk of the muscle, accompanied by a corresponding impairment of function. In by far the majority of cases the disease begins with the muscles of the upper extremity, usually the right arm, in agreement with the greater use to which the right is put in labor. According to the latest statistics of Friedreich the disease began (among 146 cases) one hundred and eleven times in the upper extremity, twenty-seven times in the lower, eight times in the lumbar muscles. Cases are very rare in which the facial muscles are first attacked (Cruveilhier), or those of the tongue (Roberts). In the latter instances there was perhaps a combination with bulbar paralysis. Sandahl found that the right upper extremity was first attacked thirty-seven times, the left upper fourteen times, and both at once eleven times. Among the muscles of the upper extremity the interossei are first invaded almost constantly; and of these the interosseus externus primus is first attacked—in my experience always so. The affection can often be diagnosticated in its earliest stages, when the wasting of the interossei is inconsiderable and the falling in of the interosseous spaces is not striking, by the diminished power of the interossei, especially the external ones; if, for example, the patient's thumb (in abduction) and his three last fingers are held, and he is told to straighten the index and then abduct it, or separate it from the middle finger, the movement is weakly performed, and the excursion of the finger is less than in the other hand, unless the disease has attacked both; fibrillary twitchings will also be easily seen in this experiment. I take pains to make this statement, because several authors (Roberts, Wachsmuth, Friedreich) make the surprising observation that when the hand is the part first attacked, the muscles of the ball of the thumb are, as a rule, the first seized upon. I have always seen the latter muscles preceded in the order of attack by the interossei, that is, by the first external interosseus. In the thumb the opponens and the weaker adductor pollicis are first and most affected, while the extensors and both abductors and flexors of the thumb remain spared for a long time, or altogether. The same is the case in the ball of the little finger. In a few cases the disease begins, not in the muscles of the hand, but in those of the shoulder, selecting from these almost exclusively the deltoid. In such instances the cause may generally be traced to an exhausting effort made with the shoulder muscles (see above); but I have once observed the deltoid attacked primarily in the person of a lady living under the most favorable circumstances, and by no means exposed to any injury of this sort. In her case the interossei were the next affected; conversely, when the interossei have been the chief seat of primary lesion, I have repeatedly found the deltoid the second point of attack, the interjacent muscles, meanwhile, remaining untouched. This typical course, and the progression by leaps, involving successively muscles and groups of muscles which are widely separated (which is certainly observed in a number of cases), are of special importance in relation to the theory of the disease.

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[1] I lately observed a case of this kind in a colleague. Pressure, experienced during the operation of version, produced at first a circumscript atrophy of the extensor carpi radialis, and afterwards an atrophy and corresponding functional debility of most of the muscles of the forearm.

Still more rarely, the disease begins with certain muscles of the upper region of the trunk (pectoralis major, serratus magnus) or with the lumbar muscles. I consider myself justified by the cases before me in saying that in those few instances where the disease is stated to have begun in the above-named pectoral muscles, the disease was not a proper classic atrophy of the muscles, but a circumscribed atrophy of certain sections of the musculature of the thorax, caused by special mechanical injuries. On the other hand, those cases where the disease began with the lower extremities, and especially with the lumbar muscles, were almost always cases of children; and in most instances, perhaps in all, its first appearance took the form, more or less clearly marked in the different muscles, of pseudo-hypertrophy, which I agree with Friedreich in regarding as merely a peculiar modification of progressive muscular atrophy, chiefly confined to childhood (see the following section). The pure progressive muscular atrophy of the adult, therefore, will be found characterized by beginning at the upper extremities and by attacking limited districts, whether the interossei or the deltoid be the first to suffer. The very rare cases in which numerous muscles are said to have been attacked simultaneously must therefore, in my opinion, be separated from common progressive muscular atrophy and be classed as a special form under the name of "multiple" or "diffuse" atrophy of the muscles, especially as these cases are characterized in their course by special malignity and acuteness, slight tendency to pauses, and a rapid and unbroken development.[1]

The emaciation of the muscles attacked, the loss of exterior contour, usually makes constant progress proportional to the growing loss of function, and produces deformities corresponding to the seat of the muscles—as, for example, the depression between the bones in disappearance of the interossei; the flattening of the ball of the thumb, and that of the little finger; the prominence of the upper end of the humerus and the acromial process of the scapula when the deltoid is affected. Yet the external loss of volume cannot always be taken as a measure of the inner atrophy of muscle; it does not always correspond with the changes in function, nor with the electric reaction hereafter to be described. A clear and perfectly satisfactory explanation of the difference has been given by Friedreich, who points out the anatomical changes upon which it is based. For, while the loss of volume can be due to progressive wasting of muscular elements and retraction of the connective tissue which proliferates in the perimysium internum, an interstitial diffuse fatty hyperplasia may occur in any stage of the disease, and especially in the early stages; earlier authors incorrectly supposed it to be a constant and characteristic factor of the muscular affection. This occurs especially in the muscles of the lower extremities, more rarely in those of the shoulder and upper arm, still more rarely in those of the hands and forearm. If there is a considerable degree of interstitial fatty hyperplasia, the volume of the muscles may remain apparently normal, in spite of a complete loss of function, and, usually, of the electric reaction as well; an excess of volume, a pseudo-hypertrophy, may even occur, as is especially the case in the muscles of the calf when the loss of substance in the upper extremity is marked. The fatty muscles in such cases are characterized by their soft, spongy, lipoma-like consistency; the functional and electric tests, and, still more certainly, the removal of the tissue by excision or the harpoon, give decisive proof. In a few cases, a well-developed panniculus adiposus may conceal even an advanced atrophy, especially in the muscles of the arm and shoulder of corpulent women.

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[1] Cf. *Friedreich*, l. c. p. 28. Case V.

A symptom of extreme frequency, both in the initial stage and during the entire course of the disease, until the muscle is wholly gone, consists in *fibrillary contractions*; that is to say, visible contractions of single bundles of fibres following each other in successive impulses, or running in waves over the surface of the muscle. Sometimes they are only single movements of lightning-like rapidity, and sometimes they form a continuous series of oscillations of greater or less intensity. Usually they appear spontaneously—that is, without visible external cause; but they are often provoked or increased by external circumstances; for instance, when such parts of the body as the shoulder and thorax, usually covered, are exposed to the air, or when active and passive movements are tried, and especially when the parts are stroked or otherwise mechanically irritated, or when electrical irritation is employed. An explanation of this symptom will be attempted in another place, in connection with the statement of the theory of the disease; for the present it is sufficient to say that its pathognomonic importance, though overrated by the first observers, certainly deserves to be rated highly, owing to the frequency and the earliness with which the symptom appears. Far less common than these so-called fibrillary (more properly "fascicular") contractions are the clonic or tonic contractions of entire muscles or groups of muscles, which also are sometimes produced or increased by external irritation, and are accompanied by distinct locomotor phenomena in the parts concerned, and by intense pain, after the analogy of the well-known cramp of the calf.

As the muscles lose their power in the progressive course of degeneration, *permanent contractures and deformities* are often developed, which present something typical in their appearance, owing to the fact that some muscles are spared while their neighbors are attacked. The case is quite analogous to that of the so-called essential paralysis of childhood, in which certain muscles or groups of muscles are usually selected, only that in childhood the conditions for the development of permanent deformities are more favorable, which insures their greater frequency. The commonest deformity is the so-called *clawed hand* (main en griffe, Klauenhand, clasped hand), which consists in permanent flexion of the last two phalanges of the fingers while they are extended at the metacarpo-phalangeal joint. This deformity is caused by atrophy of the internal and external interossei (as Duchenne first strikingly proved by faradic experiments), which, acting together, flex the fingers at the first phalanx and extend them in the second and third. When, therefore, the force of the interossei is diminished or reduced to nothing, the above effect must occur, owing to the preponderance of their antagonists, the extensor digitorum communis, the extensor indicis, and extensor digiti minimi on the one hand, and the flexor digitorum sublimis and profundus and the lumbricales on the other. The deformity is therefore pathognomonic, not so much of progressive muscular atrophy as of wasting of the interossei; I have, for example, observed it very well developed in a case of atrophy of those muscles, caused by direct traumatic lesion (compression). In consequence of the atrophy which usually occurs in certain muscles of the ball of the thumb—namely, the opponens and the adductor—there occurs a corresponding deviation of the thumb with abduction and extension in the first phalanx, and flexion in the second. In the shoulder-joint an atonic subluxation often occurs, especially in children, with dislocation of the head of the humerus in the direction of the coracoid process. A similar dislocation with hyper-extension of the arm may also appear, though more rarely, in the elbow-joint. Atrophy of the scapular and dorsal muscles may also lead to various forms of dislocation of the scapula and scoliotic or kyphotic bending of the vertebral column. Severe deformities of the lower extremity are exceptional, at least in adults, while in children the various forms of club-foot may appear, especially the paralytic pes varo-equinus caused by atrophy of the muscles of the calf and preponderance of the antagonists, tibialis anticus and posticus.

The *electrical reaction* of the affected muscles corresponds in general with the loss of volume and of voluntary power. This fact was noted by the earliest observers, as regards the faradic current. There is, therefore, no proper diminution of the electro-muscular (farado-muscular) contractility; in proportion as the contractile elements of the muscle disappear, the visible effect of direct intra-muscular faradization becomes weaker and weaker, and in extreme cases imperceptible. Some authors suppose they have observed a disproportionate loss of farado-muscular contractility; but they may easily have been deceived by the compensatory lipomatosis, which is capable of quite concealing the loss of muscular tissue.

It may be affirmed without question that the direct faradic stimulation affords the best gauge of degree of degeneration of the contractile elements, and a far exacter test than voluntary motility, or the electric motility as tested by stimulating the nerve (with faradic electricity; the "motricité" of Flourens). In both the latter tests the results are far harder to estimate; if the energy of the will is great, or if irritability is increased, or the peripheral nerves are more easily invoked to action, the effect of stimulating may be far too great; that is, it may lead to an over-estimate of the power remaining in the muscle. This is especially the case in the initial stages of the disease, in which there is often an atrophy of the integuments and an increased reflex irritability, which easily give rise to mistakes on the part of inexperienced persons. For the same reasons voluntary motility and indirect (faradic) excitability are retained longer than farado-muscular contractility, although they grow constantly less; but at the last, when degeneration is complete, and the muscles are cirrhotic and shrunken, even these are entirely powerless to provoke reaction. In all cases that I have been able to examine carefully I have seen the indirect faradic irritability disappear some time before voluntary motility, which is in perfect agreement with the action of the constant current, of which I shall presently speak.

Some authors have observed in the earlier stages an increased irritability under the action of the induced and the constant current, in direct and indirect application. Leaving out of view the sources of error, these cases (certainly very rare) must probably be referred to a temporarily heightened irritability of the intramuscular nerve-twigs, which (according to Friedreich) is due to inflammatory irritation communicated from the muscular tissue. This may also account for the prolonged closure-reaction (closure-clonus) mentioned by Benedikt.

The test of the constant current gives in general a quite analogous result. In direct galvanization the muscles, and especially the interossei, usually continue for a long time to contract in the normal way, but the force of the contraction gradually grows less, proportionally to the progress of the atrophy and the loss of voluntary motion, so that stronger and stronger currents are required to produce a minimum of contraction at closure or opening of the circuit. I have always noticed that the reaction to very strong constant currents held out longer than that to the strongest faradic currents; it is true that sometimes battery currents of sixty or more elements (Siemens) must be employed, and the current frequently closed and opened; and sometimes contraction can only be obtained by reversals of currents. It is important to establish in this way the last relics, as it were, of remaining vitality in the muscle, especially as the prognosis and treatment may in some cases be affected by the fact. The galvanic excitability of the nerve-trunks also remains unimpaired for a long time. In later stages a quantitative diminution of the reaction may occur, perhaps caused by secondary degenerative changes in the peripheral nerve-trunks; the peripheral excitability, in such cases, seems to have suffered more than the capacity to conduct voluntary impulses, as we have observed in some cases of severe peripheral, especially traumatic, lesions of the nerve-trunks. In this regard the fact observed by M. Rosenthal is of interest; the nerve-trunks behaving differently at different points in their course, so that while electric stimulation applied to a portion situated near the centre may produce normal effects, its results may be less than normal, or even quite wanting, when a more peripheral tract is stimulated. Besides these quantitative changes, qualitative alterations in the muscular reaction may attend a later period of the disease, consisting of a slight deviation from Brenner's law of contractions, namely, an increased reaction under anodic closure, and, less commonly, under cathodic opening. These phenomena, analogous as they are to certain others, point to severe degenerative changes in the peripheral nerve-trunks and muscles. Reversal of formula, and the extreme degrees of qualitative deviation from the formula of reaction have never been encountered by myself in progressive muscular atrophy.

It is proper in this place to mention one other interesting anomaly of reaction, whose frequent occurrence in progressive muscular atrophy was first remarked by Remak. I mean the so-called "*diplegic contractions.*" Remak found that contractions could be produced in the atrophied muscles of the arm, when the positive electrode was placed in an "irritable zone," which extends from the first to the fifth cervical vertebra, or, still better, in the carotid fossa or the triangle between the lower jaw and the external ear, while the negative was put below the fifth cervical vertebra. The contractions were always upon the side opposite to the anode, but when the electrodes were applied in the median line they occurred on both sides; if the current was very weak, they were limited to the muscles most severely affected. Remak regarded these as reflex contractions, originating from the superior cervical ganglion of the sympathetic, and especially as the patient perceived a sensation behind the ball of the eye when the current was closed; he made some extensive theoretic conclusions, about which we shall have to speak later. While Fieber and Benedikt could not produce the diplegic contractions, Meyer, Drissen, and Erb (in a case reported by Friedreich[1]) confirmed Remak's statement. For my part, in spite of regular attempts, I have succeeded in demonstrating the diplegic contractions in only one case of progressive muscular atrophy, and here the phenomena by no means followed the order as stated by Remak. I also entirely disagree with Remak as regards the interpretation of this symptom, and believe that they ought to be taken as genuine reflex contractions, independent of the sympathetic, and caused either by excessive irritability of the central reflex apparatus, or by an abnormal excitability of the muscles themselves.[1]

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[1] Case V. of *Friedreich's*, above quoted.

Among those symptoms which occupy a secondary place in respect to frequency and dignity, the *disturbances of sensibility*, the *vaso-motor* and *trophic disturbances*, and the *oculo-pupillary* phenomena deserve a special consideration.

The *sensibility*, in the majority of cases, presents no change worth mentioning. But in a certain number of cases the development of the affection in the muscles, or at least its higher grades of development, are preceded by paroxysms of pain in the affected parts. Sometimes the pains follow the course and distribution of single nerve-trunks, as that of the median and ulnar nerves in the arm; sometimes this is not recognizable, and the pains seem rather to take their origin in the muscles themselves, in the sensitive nerves which supply muscular tissue. The latter explanation is at least favored by the circumstance that compression and active or passive motion provoke or increase the pain, as also, that electro-muscular sensibility seems to be increased in a few cases. In later stages, after the neuralgic and hyperalgic symptoms have passed away, or even while they still continue, a moderate degree of anæsthesia is often developed in the form of *partial paralysis of sensation* (dulling of common sensation, analgesia with normal sense of touch[2]), especially in the finger-tips and hands. The farado-cutaneous sensibility may present a similar diminution. I have never observed higher degrees of loss of sensibility in cases of uncomplicated progressive muscular atrophy. Paralgic symptoms, as a sensation of cold and numbness in the finger-tips, formication, etc., are more common. Finally, the exaltation of reflex excitability must be mentioned here; for, independently of the diplegic contractions, which probably are due to this cause, abnormal reflexions may occur (*e. g.*, when needle-pricks, tickling of the soles, etc., are employed), especially in the initial stage of progressive muscular atrophy.

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[1] Cf. Centralblatt. 1868. No. 3.

[2] Cf. *Landois* and *Mosler*, Neuropatholog. Studien Berl. klin. Wochenschrift. 1868. No. 45.

Vaso-motor trophic disturbances of various degrees and extent may occur in the affected regions. The *temperature* of the limbs that have been attacked is sometimes a little elevated in the beginning; Baerwinkel demonstrated in one case a local elevation of 1° with a full radial pulse; Frommann found on the side first attacked a rise of 0.2° or 0.3° C. In most cases, and in advanced stages, a local rise of temperature cannot be demonstrated, and at a later period a decided lowering may occur, which, according to M. Rosenthal, may amount to 4° C. The *color* of the parts is usually normal; sometimes it is pale, and rarely of a distinctly livid or cyanotic character. In a few cases a local ischæmia is observed from time to time, especially of the hands and fingers, a sudden coldness and paleness of the parts, followed by relaxation of the vessels, redness, etc., such as occurs in the well-known forms of vaso-motor neurosis. It is also worthy of note that in a few cases *excessive local sweatings* occur, which usually seem to pass into a general increase of the secretion of sweat.

Of the *trophic* disturbances (in the stricter sense of the term), an *atrophy of the outer integuments* must be mentioned, which often accompanies the muscular lesion. It affects all the layers of the integument, the epidermoidal tissues as well as the cutis and the subcutaneous tissue, but scarcely ever reaches an advanced degree, even when the muscular disease is far advanced, and may be wholly wanting. Here perhaps we ought also to mention the *painful swellings of the joints* (*arthritis nodosa*), first spoken of by Remak, and observed chiefly in the early stages of the atrophy; Remak conceived of them as "neuroparalytic inflammations," and connected them with the sympathetic. I have observed them in some of the phalangeal joints in several cases, which were treated early; likewise in one case of primary disease of the right deltoid where the corresponding shoulder-joint was affected in a pronounced manner. These joint-affections are doubtless genetically related to the "neurotrophic" arthropathies of tabes dorsalis, of which Charcot[1] has spoken, except that the latter are more frequent in the large joints, as the knee, shoulder, elbow, etc., and therefore are of greater pathological importance.

Oculo-pupillary symptoms, of the sort previously described under Hemicrania and Basedow's disease, occur comparatively rarely in progressive muscular atrophy, but they deserve a special attention on account of their theoretic relations.

Contraction of the pupil was mentioned by Schneevogt (1855) and Baerwinkel (1858). The first exact description originates with Voisin, and relates to a case in Bouillard's clinic. The patient was a man, aged forty-four, and had suffered seven or eight years with the disease—first in the left, then in the right arm. His left eye was the first attacked; a contraction of the pupil appeared, which reduced it to half the size of the other, but did not prevent its reacting normally; the cornea was flattened, so that its most prominent portion seemed nearer by one millimetre to the iris. In a few months the same phenomena occurred in the right eye; both pupils were equally contracted, and reacted slowly, the cornea was equally flattened on both sides, and the sight of both eyes was poor. In a case given by Menjaud, of muscular atrophy chiefly confined to the distribution of the median and ulnar nerves of both arms, there existed a considerable contraction of the left pupil. Bergmann also observed in one case contraction and slow reaction of the left pupil; M. Rosenthal found unilateral contraction in four cases; Friedreich found in one temporary contraction and sluggish reaction of both pupils. These few positive observations are far outnumbered by the negative; for example, Duchenne, in remarking on Voisin's account, says expressly that among several hundred (!) cases he never once met with the phenomenon. And in all those which I have examined, I have never been able to make out these disturbances of innervation of the eye. They must, therefore, be looked upon as a very rare complication of compressive muscular atrophy.

Of the *general symptoms*, *fever* must first be mentioned; it may occur in a few cases of progressive muscular atrophy, especially in the initial stage, either as repeated attacks of chill or as a continuous increase of temperature, very slight in amount, which lasts for days or even months. Remak is, as far as I know, the first who has called attention to the existence of febrile symptoms in connection with arthritis nodosa in the initial stage of the disease; he drew from its presence a fresh proof of the connection he assumed to exist with the sympathetic nerve or the sympathetic centres in the spinal cord. It remains for further observation to decide whether these initial symptoms of fever are most common in the cases which are accompanied by acute arthropathy, and whether they perhaps are connected with the latter. In the later stages there may occur transitory or permanent elevations of temperature, which may be variously interpreted ; they are usually dependent on complications (diseases of the lungs, decubitus, etc.), but in a few cases they seem essentially to depend on the pyrogenic action of products of inflammation and decomposition, reabsorbed from the muscles and infecting the blood (Friedreich).

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[1] Archives de physiologie. I. 1868. II. 1869.

Friedreich mentions the case of a man of twenty-eight years, whose pulse usually exceeded 120, who suffered from elevation of temperature that lasted continuously over seven months, striking pallor of the face and the mucous membranes, frequent dryness of the tongue, disposition to diarrhœa, constant and abundant excretion of urates, and a considerable swelling of the spleen. The final result could not be known. No similar cases have as yet been reported.

Among the general symptoms may be mentioned the *changes in the secretions of the sweat and urine* which have been observed in some cases. An excessive sweating (hyperidrosis) of a generalized character occurs especially in the later stages (Frommann, Friedreich) ; its origin and its relation to the other symptoms are at present wholly uncertain (see "Analysis of Symptoms.") Concerning the qualitative and quantitative changes in the composition of the urine, there are but few and very discordant statements. Friedberg and Frommann each observed in one case a deposit of lime in the urine ; the latter found the urine pale, turbid, slightly alkaline, with abundant sediments of carbonate of lime, which he believed to originate from the atrophied muscles. Bamberger, however, found in one case urea and chloride of sodium in normal or increased quantity, great increase of sulphuric acid, and considerable diminution of the uric and phosphoric acids. An examination of the blood at the same time showed a considerable increase in albumen and blood-corpuscles. More important, but not yet confirmed by other observations, is the increase of creatinin in the urine, observed by M. Rosenthal in three cases. Friedreich found the urine acid in three cases, of quite high specific gravity, containing more or less constant sediments of urates, and free from albumen. It is greatly to be wished that the number of exact and full examinations of urine might be increased, with the view of explaining the chemical changes in muscular substance produced by the disease.

In passing now to the consideration of the progress of the disease, we have, in the first place, to make a few remarks upon the *manner in which it extends itself*, and the *irradiation of the myopathic process*. This important point, unfortunately, is not yet so clearly made out by clinical observation as might be desired. We have seen that the disease begins most often with certain muscles of the hand (interossei, ball of the thumb), more rarely with the shoulder (deltoid), still more rarely with the lower extremities (lumbar muscles) or the muscles of the trunk. In the ordinary course of the disease, when the upper extremities are primarily or exclusively affected, a series of muscles belonging to the forearm, upper arm, and region of the shoulder, are gradually implicated. The influence of contiguity in the progress of the disease, a continuous passage from affected muscles to their neighbors, a direct propagation of the inflammatory (myositic) process, due to propinquity, can by no means, in my opinion, be certainly shown. It is contradicted by the circumstance already mentioned, that the primary affection of the interossei is very often followed directly by that of the deltoid, and *vice versa*, the intervening muscles remaining intact. The adherents of the myopathic theory, it is true, help themselves out with the assumption that two centres of disease may exist at once—in the muscles of the hand and in the deltoid. They describe the disease as ascending from the former to the forearm, and as descending from the latter to the upper arm, and crossing laterally to the muscles of the chest and back.[1] The case is similar, according to them, in the lower extremities, where the process usually ascends from the legs to the thighs, buttocks, and loins, or from the loins to the higher muscles of the trunk, and seldom descends from those of the buttocks and loins to the lower extremities. It is true, it is just as easy to

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[1] Cf. *Friedreich*, l. c. p. 230, and elsewhere.

admit a still greater plurality of centres of this sort, one for each single muscle attacked, as it is to grant several centres which gradually unite in one. But, leaving the question of propagation by contiguity unsettled for the present, there remain two other important points in respect to the progression of the muscular disease. One relates to the way in which the process is limited, especially to segments of the skeleton marked by the greater joints; the other, to the habitual escape of certain muscles and groups of muscles. In a large number of cases the disease, beginning with the muscles of the hand, does not pass beyond the wrist, or, at least, remains comparatively stationary for a very long time; in other cases another series of muscles in the forearm, especially the extensors, are affected, and a bound seems set to the progress of the disease at the elbow-joint. Conversely, when the disease begins at the shoulder, the muscles of the upper arm only are often attacked, while those of the forearm are spared. In atrophy of the leg the knee-joint seems to set an upward limit to its progress. It is true, as Friedreich states, that this bound can be passed, and, after the process has overleaped the joint, it may attack the neighboring limb, and there make more or less progress. But in this case also, according to Friedreich's view (which is supported by several autopsies), the spreading takes place entirely *per contiguitatem*, *e. g.*, from the biceps and brachialis anticus to the body of the supinator longus, which lies directly upon their insertions; also to the extensor carpi radialis longior and brevior, and the upper part of the extensor digitorum communis, which lies next to the latter. Besides, the temporary or permanent arrest by no means always takes place at the joints, but often enough occurs before the disease reaches them, as may occur at any point whatever of the trunk. There are even cases in which the disease arrests itself in the body of a muscle, so that only a few larger or smaller segments of muscular bundles are affected, while the rest are perfectly sound. Friedreich describes such a condition in the cucullaris, deltoid, and the glutæi. It is remarkable that all these muscles possess a double or even a more numerous supply of nerves. As respects the relative or absolute immunity of certain muscles, those of the neck and head are affected only in exceptional cases. The instances where some muscles of the head, especially those of the lips and tongue, are affected, usually belong to a combination with progressive bulbar paralysis, or to progressive glosso-labial atrophy, which is still distinguished by some as an independent disease. The striped muscles of the organs of the higher senses (extrinsic muscles of the globe and internal muscles of the ear) seem never to be affected. The diaphragm, the straight muscles of the abdomen, and those of the larynx, are unquestionably affected in some cases, though rather rarely.

It remains to say that the symmetric muscles of both halves of the body are usually affected, but by no means to the same degree and extent. Often, even after a long continuance of the disease, its intensity is much less in one extremity than in the other; and in these cases the peculiar condition of a crossed attack is often developed, in which one upper and the opposite lower extremity are chiefly affected. I have elsewhere described several very marked cases of this sort, some of them in connection with pseudo-hypertrophy of the lower extremities.[1]

With the exception of certain cases already described, the *course* of the disease is generally very protracted. Its progress is usually extremely slow, especially at the first, and produces at first no trouble, and particularly involves no danger to the general health or to life; its results are confined to the local disturbances caused by the increasing disability of motion. A spontaneous retrogression of the process in the muscles after they are once atrophied, with a spontaneous disappearance of the clinical symptoms which depend upon the atrophy, never takes place, as far as we can judge. But there is no doubt that the disease may remain stationary for a longer or shorter time, even for years, at any period of its progress; this has been shown by what has been said above. Can this arrest continue, not only for years, but permanently—that is, so that there will be no relapse during the normal period of life? We are hardly entitled to answer this question in the affirmative with the experience we have had, and especially when we reflect that our knowledge of the disease does not extent beyond twenty-four years in all.

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[1] Virchow's Archives, Vol. 49. p. 446; vol. 53. p. 361.

The first serious danger to life in progressive muscular atrophy occurs when the respiratory muscles are attacked; though the most important of these, namely, the diaphragm and the muscles of the larynx, as well as the recti abdominis, are usually spared. Yet the very frequent affection of the large accessory muscles of breathing, as the pectoralis major, serratus anticus magnus, cucullaris, etc., may be an indirect source of danger; slight diseases of the respiratory apparatus, impediments to breathing which would otherwise be easily overcome, such as simple catarrh of the bronchi, may, under some circumstances, produce asphyxia and death. In other cases death is caused by the rapid progress of bed-sores, during which the patient, especially when the lower extremities and the muscles of the trunk are affected, grows more and more helpless, cannot leave his bed, cannot even change his position without help, which circumstance, especially if the muscular layer of the back be atrophied or the integuments thinned, exposes him very greatly to the danger of bed-sores.

A cause of death which does not lie in the disease itself consists in the complications which may be allied to the disease. One of these is so frequent, and is clinically so nearly allied to progressive muscular atrophy, that it must be considered not as an accidental combination, but as a combined disease, arising from the same or similar pathogenic causes. This is progressive bulbar paralysis. We shall speak further of its relation to progressive muscular atrophy, when we come to the theoretical discussion of the latter; at present let it be simply remarked that it may form a cause of death, either by producing a gradual palsy of the muscles of swallowing and of the larynx, or, when it occurs in the apoplectic form (Joffroy), it may be accompanied by hemorrhage in the rhomboid fossa, or by embolism or thrombosis of the vertebral artery, with immediate palsy of the respiratory centres. In many cases death has been caused by acute intercurrent affections of the lungs or chronic miliary tuberculosis, either while the disease is in full progress or after it appears to have been arrested.

Pathological Anatomy.

We must distinguish the anatomical changes in the *muscular apparatus* from those in various parts of the *nervous system*. We begin with the former, partly because the conditions themselves are simpler and more constant, partly because their interpretation is clearer than is the case with the alterations in the nervous system.

We have information regarding the diseased muscle in the living subject, derived from excision or from explorative puncture with Middeldorpff's harpoon, Duchenne's emporte-pièce histologique, or similar instruments. This procedure has been less used in progressive muscular atrophy than in pseudo-hypertrophy, which is probably due to the fact that the introduction of the trocar into a hyper-voluminous muscle seems easier of execution than when there is an atrophied mass to deal with. Excision of muscle is dangerous, and ought decidedly to be rejected; it leads, as I have convinced myself in a case recently operated upon by another, to a long-continued suppuration, even if the wound at first behaves very well; extensive erysipelas and severe general symptoms have also been observed (Friedreich). Puncture is free from these dangers, but does not always lead to certain results, partly because the muscle is not penetrated, and partly because the portion removed may happen to be unaffected; it must, therefore, in many cases, be repeated.

While most of the earlier investigators, especially Meryon, Duchenne, Cruveilhier, Wachsmuth, Valentiner, and others, regarded the process as simple fatty degeneration of the muscular fibres with secondary disappearance, and finally a perishing of the sarcolemma, others, as Robin, Friedberg, Foerster, Schueppel and Hayem, defended more or less decidedly the inflammatory theory, considering the fatty metamorphosis of the primitive fibres as secondary and subordinate. The results of Charcot and Joffroy, Duménil, and others, are less definite. Recently these questions have been brought to a conclusion by the investigations of Friedreich, which I think fully exhaustive and convincing upon this point. According to these, the disease consists in an essentially inflammatory process, a "polymyositis chronica progressiva." The first changes begin in the perimysium internum as hyperplastic growth of the interstitial connective tissue in its finest ramifications among the single primitive bundles. At the same time there occur phenomena of irritation in a greater or smaller number of primitive bundles, in the form of swelling and multiplication of the muscular corpuscles, and especially proliferations of their nuclei, and sometimes parenchymatous granular cloudiness of the cross-striped fibrillary substance. In a few cases hypertrophied muscular fibres, and a dichotomous or trichotomous division of the hypertrophied fibres (Friedreich), was seen. During this increase in interstitial tissue, the wasting of the muscular substance goes on in various ways, partly by simple emaciation and progressive dissolution without loss of transverse striation (sometimes preceded by longitudinal, transverse, or elementary fission), and partly by waxy or fatty degeneration. The final result is a more or less complete *fibrous degeneration* (*cirrhosis*) of the muscle. An accessory process, by no means constant or essential, is the diffused lipomatosis of the muscle, which appears sometimes early, sometimes late in the course of the disease.

It is important to know that the development of fat, where present, always begins outside of the proper muscular elements, within the hyperplastic interstitial connective tissue. In the early stages it is easy to show the process of origination of fat-cells from the corpuscles of the connective tissue, by the latter becoming filled with larger or smaller drops of fat which run together into globules, as Virchow has described it. If the lipomatosis occurs at a stage when the atrophy of the muscular elements proper has not yet reached a great degree of development, we can see yellowish streaks and lines of fatty tissue through the muscle, which in other respects looks normal. In the highest degree of fibrous degeneration the muscles, according to their original shape, assume the form of thin, hard, reddish gray cords, or else of tendinous membranes, in which light reddish stripes and islands are seen, the relics of the old muscular tissue. But if a considerable development of fat-cells subsequently occurs within the already cirrhotic muscle, its volume may increase again, so that it regains or overpasses its former dimensions, as is especially the case in the calf of the leg; but a section of an extreme case of this lipomatosis shows the muscle changed into a mass of fatty tissue, which exhibits the original fibrous structure of the muscle and the course of the former bundles of fibres by the direction of the streaks of fat; the remains of muscular substance are represented by a few reddish, gelatinously transparent streaks in the lipomatous tissue. The cause of the lipomatosis is at present quite obscure, but it must be referred to local peculiarities of nutrition, rather than to general characteristics, as its predilection for certain muscles proves. In regard to the other anatomical details the reader must be referred to Friedreich's complete account.[1]

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[1] L. c. chap. 2. pp. 46–92.

The statements of different authors regarding the pathologico-anatomical changes in the nervous system unfortunately disagree widely. This is especially true of the older accounts, and is explained partly by the defectiveness of the old methods of investigation, and partly by the circumstance that, being prepossessed for one or another view, investigators have turned attention sometimes to one, sometimes to another section of the nervous system. The *anterior spinal nerve-roots* were the first to assume importance, owing to Cruveilhier's view; Cruveilhier himself found a high degree of change in them in two cases.

The first of these was the case of the rope-dancer Lecomte, which was mentioned in the introduction. The brain and cord and posterior roots were normal, but the anterior roots from their point of exit to where they unite with the posterior were greatly atrophied. The muscular branches proceeding from these trunks were smaller in size than the cutaneous, and in them the neurilemma preponderated over the muscular substance. In those nerves which ramify in the ball of the thumb scarcely anything but neurilemma existed. In the second case the anterior roots were found similarly atrophied, by comparison with the posterior; the ratio of their thicknesses in the cervical region was 1 : 10 (normally 1 : 3); in the dorsal and lumbar regions as 1 : 5 (normally 1 : 1½ or 2). The posterior roots, brain and cord were unchanged in this case also.

Atrophy of the anterior spinal roots was confirmed by the following authorities; in some cases it is mentioned in connection with other changes: Reade, Duménil (two cases), Schneevogt, Valentiner, Menjaud, Clarke (three cases), Trousseau, Vulpian (two cases), Luys, Jaccoud (two cases), Schueppel, Grimm, von Recklinghausen, M. Rosenthal, Hayem, Charcot and Joffroy, Baudrimont, Friedreich (two cases). On the other hand, the integrity of the anterior spinal roots is expressly stated by Oppenheimer, Axenfeld, Aran (two cases), Cohn, Friedberg, Meryon, Virchow, Clarke (in the case observed with Gairdner), Frommann, Gull, Friedreich (four cases), Tuerck, von Recklinghausen (second case from Bamberger's Clinic), Joffroy, and Frerichs (case described by Swarzenski). Thus twenty-six positive observations stand opposed to nineteen negative. In a few reports of autopsies, not here given, the anterior roots receive no special mention.

While Cruveilhier had found the *spinal cord* itself uninjured, Valentiner, on the contrary, discovered in 1855 a central softening of the medulla in the neighborhood of the three lowest cervical and the uppermost dorsal nerves. The elements of the region of transition from gray to white substance were obliterated; the softened places showed numerous granule-cells. Schneevogt found also, in addition to the lesions of the sympathetic, of which more will be said presently, a softening of the cord from the fifth cervical to the second dorsal nerves. Frommann, in the case observed in Leubuscher's Clinic, describes as the leading alteration a red softening, involving chiefly the *anterior and lateral columns* from the medulla oblongata downward; it involved especially the commissures and the innermost parts of the anterior columns, lying next to the commissure. Luys seems to have been the first to call attention to the frequency of the affection in the *gray substance;* he found the latter, in the neighborhood of the cervical enlargement, full of very hyperæmic vessels, which were studded and surrounded with granular exudation (fat-granule cells). The same granular masses, and many corpora amylacea, were scattered through the gray substance. *The ganglion-cells of the anterior cornua had almost disappeared in the part affected, and were replaced by those granular masses;* only here and there could a few ganglion-cells be recognized, in a state of decided retrogressive metamorphosis, brownish in color, full of dark granules, and with their anastomoses and processes broken off or entirely wanting. The degeneration affected principally the left anterior cornu, corresponding with the seat of the muscular atrophy, which was chiefly on the left side, and the left-sided atrophy of the anterior roots.

This observation of Luys furnishes an important contribution to the changes of the gray substance, especially of the anterior cornua. For further developments of this point we may especially refer to the numerous and careful examinations by Lockhart Clarke. In all the six cases described by him, more or less extensive changes in the gray substance were found, with especial implication of the anterior ganglion-cells, and in part with changes in the white columns and the spinal roots. The changes consisted partly in great dilatation and crowding of the blood-vessels, partly in gray softening with fatty granular deposits ("granular disintegration"), which must clearly be regarded as the result of *irritative inflammatory* processes, and partly in induration with abundant hyperplasia of connective tissue and atrophy of the nerve-elements. Especially important are the changes (observed previously by Luys) in the *multipolar ganglion-cells of the anterior cornua*, in the description of which Clarke agrees entirely with other observers (Duménil, Schueppel, Hayem, Charcot, and Joffroy): brownish discoloration, granulation, thinning and loss of the processes, diminution of the size of the cell-body, and its transformation into non-nucleated angular bodies, which sometimes glisten, or possess indistinct boundaries, with final complete disappearance of the cell. The cause of this change is probably without doubt due to the inflammatory softening or hardening of the gray substance of the anterior cornua, in cases where such inflammatory changes are found. Whether in other cases, as Friedreich supposes, the atrophy of the ganglia is possibly due to the suspension of the physical function of the muscle for years, may remain an open question.

A disease confined chiefly or wholly to the gray substance is found noted in another series of autopsies. Gull found, as the leading change, a considerable dilatation of the spinal canal in the cervical region, between the fifth cervical vertebra and the origin of the third and fourth dorsal nerves. The cavity thus formed was full of a serous fluid; the gray substance had disappeared, with the exception of a thin layer which surrounded the cavity and could be stripped off like a membrane; the white medullary striæ and the roots seemed normal. The observations of Schueppel are very similar, except that the hydromyelia extended down to the tenth dorsal vertebra. Hayem also found the gray matter of the cervical region exclusively affected (indurated), especially in the anterior cornua, while the white columns were quite normal. Grimm found in his case a considerable enlargement of the central canal at the expense of the gray substance, which was flattened by pressure to a ring-shaped plate. The cord also presented in its lower cervical and upper dorsal regions a fusiform swelling, composed of a new formation (medullary sarcoma). Upon microscopical examination the gray substance appeared composed of fine connective tissue with its fibres much interlaced, amongst which a few nerve-fibres and some atrophied nerve-cells were visible. In the white substance there was hyperplasia of the connective tissue along with increase of the axis cylinders. The nerve-roots were in fatty degeneration, especially the finer fibres of the anterior roots. Grimm considers that this observation allows the hypothesis of a chronic myelitis, as the cause of the lesions found; he thinks the carcinoma an accidental complication, which commenced at a later period.

While these observers found the gray substance the chief, if not the only seat of alteration, there are not wanting cases in which there was nothing found but a *degenerative atrophy of the white columns* of the cord; this affected sometimes the *antero-lateral columns* (as in Frommann's case), and sometimes the *posterior*. Considerable atrophy of the antero-lateral columns was found by Baudrimont, Duménil (and others), though Duménil found it conjoined with hyperæmic inflammatory changes in the gray substance and atrophy of the ganglion-cells. Virchow was the first to discover in the posterior columns gray degeneration, visible to the naked eye, with extensive loss of nerve-substance, deposition of a soft substance with numberless corpora amylacea and longish oval granulated nuclei. The very same change was observed by Friedreich in one of his six cases; and in one of Clarke's cases, along with disease of the antero-lateral columns and the gray substance, there was a preponderant change in the posterior columns; and in the case of Swarzenski's from Frerichs' Clinic a total transformation of the latter was found, with granular deposits. For the sake of completeness let it be added that in a few cases the posterior cornua (Joffroy), and the posterior roots (Menjaud, Clarke, Duménil), and the inter-vertebral ganglia (Duménil), were found in a state of degeneration, though this was not confined to them.

These positive observations upon the spinal cord are met by a number of negative ones, in which the organ, including the spinal roots, exhibited no trace of pathological change. In this class must be mentioned the older cases of Landry, Oppenheimer, Meryon, Tuerck, Cohn, Friedberg; one case by Malmsten; three cases which are said by Duchenne[1] to have been examined post-mortem in the Paris hospitals (two by Aran, one by Axenfeld); one from Bamberger's Clinic, and four by Friedreich. My summary of the cases gives a total of 34 decidedly positive statements regarding the spinal cord, against 15 negative, which stand recorded in literature. The attempt to base any statistical conclusions upon the present most incomplete and defective material may be considered as wholly premature. The old proverb, "Non numerandæ, sed perpendendæ sunt voces" is certainly applicable here, if anywhere.

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[1] Électrisation localisée. 2d éd. 1861. p. 520.

We have to speak, finally, of the changes found in the *sympathetic* and the *peripheral nerves.* Schneevogt seems to have been the first to direct his attention to the sympathetic; he found (in a case which is further remarkable for the contraction of the pupil) partial softening of the cord and atrophy of the anterior roots, with a great amount of destructive change in the cervical sympathetic; the latter was almost converted into a fatty cord, in which the nerves were pressed aside by interjacent fat-cells with beautiful crystals; the cervical ganglia were almost wholly changed into fat-cells, and the thoracic part of the sympathetic was also somewhat abundant in fat. Jaccoud further found in two cases (men aged fifty-seven, in Béhier's department) an atrophy of the anterior cervical roots, and with it a fibrous fatty degeneration of the sympathetic, which had made very great progress. The trunk of the cervical sympathetic was absolutely changed into fibrous connective tissue, manifestly of very old date, in which large deposits of fat occurred in places; in consequence, the nerve-tubes were in a condition of secondary atrophy, and that in a still higher degree than the spinal roots. The uppermost cervical ganglion showed what seemed to be the first stage of the process—a considerable hyperplasia of the cortical and interstitial connective tissue, but without atrophy of the nervous elements. The rami communicantes exhibited an atrophy which corresponded with the condition of the anterior roots; the median nerves contained a large number of healthy fibres, with a few in a state of pathological change (simple atrophy, disappearance of the medullary sheath, and even of the axis-cylinder). Jaccoud infers from the age of the various processes that the disease unquestionably began in the cervical sympathetic, and spread from thence both centripetally (rami communicantes, anterior roots) and centrifugally (median nerves).

Changes in the sympathetic are also mentioned by Swarzenski (namely, thinning of its trunk and the two uppermost ganglia) and by Duménil (advanced fibrous fatty change of the cervical and thoracic portion; abundant hyperplasia of connective tissue; disappearance of nerve-fibres and regressive metamorphosis of the ganglion-cells). In the majority of cases no examination of the sympathetic was probably made, but a certain number of good observers testify to its integrity, as Landry, Frommann, Menjaud, Hayem, Charcot and Joffroy, Duménil (3 cases), and Friedreich (6 cases). In all, therefore, there are 5 positive observations, to which 14 negative are opposed. It is also important that Duménil, in the one case of sympathetic degeneration observed by him, found also an advanced fatty degeneration of the spinal ganglia, and of the anterior and posterior spinal nerve-roots in the region of the neck.

In regard to the peripheral nerves, the great majority of observers have reached positive results similar to those of Cruveilhier and Jaccoud, of which we have spoken; thus, Schneevogt (nervus ulnaris), Trousseau (n. axillaris, medianus, radialis, ulnaris), Virchow, Friedberg, Hayem, Charcot and Joffroy, von Bamberger, M. Rosenthal, Friedreich, and others. The degenerations were sometimes confined to the finer intra-muscular nerve-branches (Friedberg, von Bamberger); sometimes they existed in the larger trunks also, and even in the plexuses (Rosenthal). They mostly bore the character of active irritative processes, with hyperplasia and nuclear proliferation of the outer and inner neurilemma, multiplication of nuclei, and fibrillary thickening of the sheath of Schwann (perineuritis and neuritis interstitialis chronica); there was also swelling and varicose dilatation of the medullary sheath (Friedreich), with secondary atrophy of the nerve-fibres, which in some cases were quite extinct. It may be that in these cases also, as Friedreich supposes, the atrophy of the nerve-elements is favored by their permanent inactivity. In a few cases (Duménil, Friedreich) examination of the peripheral nerves gave entirely negative results.

Theory of the Disease and Analysis of the Symptoms.

We cannot attempt in this place to state, much less fully to disprove, all the theories which have arisen in the course of time in relation to the nature of progressive muscular atrophy. Most of them dispose of themselves after a little reflection upon the preceding clinical and pathologico-anatomical facts. Thus, the theory of Cruveilhier, though ingeniously constructed, hardly requires a serious refutation at the present day; and the views of authors who placed the origin of the disease in the peripheral distributions, in the antero-lateral columns, or even in the posterior columns of the cord, though apparently supported by single observations, may be passed over in simple silence. In regard to the assumption indicated by Schneevogt, and more fully developed by Remak, Jaccoud, J. Simon, and others, that the primary affection is in the sympathetic, I have already elsewhere[1] expressed the opinion, after a thorough criticism of the facts, that neither our knowledge of the anatomical facts nor our physiological analysis of the symptoms places us in a position to form a final opinion regarding the rôle of the sympathetic in the disease in question. At that time I formulated the questions: "Is the sympathetic regularly affected in these cases?—and if it be, does it become affected by centripetal propagation of the primary muscular disease to the peripheral nerves, the roots of the spinal nerves, and the rami communicantes? Or, *vice versa*, is the sympathetic the first to be affected, and does the disease extend, on the one hand, centrifugally to the peripheral nerve-trunks and the muscles, and, on the other, centripetally to the spinal centres of the sympathetic fibres involved?" The first and principal question I now think it necessary to answer definitely in the negative, in view of the considerable number of decidedly negative observations presented by thorough observers. With this question the second alternative also falls—its only support has consisted of the statements of Jaccoud; and the first alternative is at least very much restricted by the large number of cases in which changes of the peripheral nerves and the anterior roots were found, without a secondary lesion of the sympathetic, and without a lesion of the rami communicantes (Duménil). I therefore can only regard the affection of the sympathetic, which is unquestionably present in some cases of progressive muscular atrophy, as a *combined affection*, characterized clinically by its special group of symptoms (oculo-pupillary phenomena, etc.), after the analogy of the explanation, which, I believe, we must assign to progressive bulbar paralysis when found combined with progressive muscular atrophy. As in the latter a transmission of the fundamental pathologico-anatomical change takes place from the cervical cord, or *vice versa*, so in the former there is a diffusion through the rami communicantes to the cervical and dorsal portion of the sympathetic trunk and ganglia.

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[1] Pathologie des Sympathicus. pp. 96, 97.

There remain, therefore, for discussion, only two theories of the disease, namely, the *myopathic*, which has remained essentially unchanged since Aran, and the *neuropathic*, in the form which it has lately assumed in consequence of the investigations of Lockhart Clarke, Hayem, Luys, Charcot, and others, according to which progressive muscular atrophy is essentially due to *primary alterations in the ganglion-cells of the anterior gray cornua*, the latter appearing to stand in a relation to the nutrition of the muscles which is as yet obscure and not even directly proved.

Those who cling to the myopathic theory can no longer, of course, deny the frequency of pathologico-anatomical changes in the nervous apparatus, and especially in the cord; they can only attempt to present the muscular lesion as primary and the nervous lesions as secondary, dependent on the former. This is the final statement of the criticism which Friedreich has lately carried out against the neurotic theories. Friedreich formulates the mutual relation in the following sentences: "Progressive muscular atrophy, beginning as a primary chronic myositis, is capable of producing secondary disturbances of the nervous system, consisting in a chronic neuritis which attacks the intra-muscular nerves and ascends along the course of the nerve-trunks to the roots of the nerves. The neuritis may further attack the spinal cord itself, producing chronic myelitic processes, which are capable of extending in various directions in the cord. The inflammation which is propagated within the nerve-routes may come to a halt at any point in its course, and the extent to which the disturbance of the nervous apparatus reaches is essentially dependent upon the degree of activity of the changes in the tissue which are taking place in the muscle and furnish the source of irritation. Besides this, certain regressive disturbances of nutrition of the peripheral nerve-fibres, and of the ganglion-cells of the gray anterior cornua of the cord, are to be considered as the results of disturbed motor function."

Without desiring to express a decided opinion—for I consider the question not yet ripe, not even since Friedreich's work—I wish nevertheless to call attention to a few deficiencies which I think still exist in Friedreich's demonstration. When Friedreich adds, after the words we have just quoted, "only by this theory can the great variety of morbid conditions found in the nervous system in single cases be naturally explained," it is hard to understand him; we should expect, on the contrary, in the case of an inflammatory process which begins at the periphery and travels, by pre-existing paths, in a centripetal direction, to find a most constant and unvarying set of changes in the said nervous organs. If, moreover, the lesions of the cord are to be regarded as the final results of an inflammatory process continuously ascending in the nerve-paths, it should be assumed that in all cases, where degenerative changes of the gray substance, especially of the anterior cornua, are present, there will be demonstrable changes also in the peripheral nerve-trunks, the plexuses, and the anterior spinal roots. But this is so far from being the case that Friedreich's neglect to see this circumstance, or the slight importance he attached to it in connection with the theory, cannot but surprise us. I will simply call to mind cases, such as for instance that described by Clarke and Gairdner, in which there was an advanced myelitic softening in the cervical part and down to the third dorsal nerve, while the nerve-roots showed no sign of disease; or the case by Gull, previously cited, where there was hydromyelia with disappearance of the gray substance, without changes in the nerve-roots. Among the nineteen cases above mentioned, in which there was no change at all in the anterior spinal roots, there are several in which changes of the spinal cord, sometimes extreme, were present (Virchow, Frommann, Joffroy, Frerichs, and others). It is of course impossible to say that these cases were insufficiently investigated; we have only to give up the attempt to explain them according to a programme. It is hard to make those other cases agree with Friedreich's theory, in which there were no changes, or but very slight ones, in the peripheral nerve-trunks, while there was advanced atrophy of the anterior roots, and in some cases great alterations of the spinal cord. The theory, frequently resorted to by Friedreich, which makes the suspension of muscular action (originating in an affection of the muscles), slowly and incompletely as it is accomplished, to be of and by itself the cause of regressive disturbances of nutrition in the peripheral nerve-fibres, and the ganglion-cells of the anterior cornua, I cannot regard as justified; neither physiological facts, nor those of experimental pathology, nor clinical evidence, give a sound support to this assumption.

The arguments drawn by Friedreich from the manner of progression of the muscular affection and from etiology, I am compelled to regard as equally far from conclusive. The etiological facts are at least thoroughly ambiguous, and bear as much upon the nervous system as upon the muscles—*e. g.*, the influence of congenital diathesis, over-exertion, traumatic lesions, etc. Certain facts also, connected with the manner of extension of the disease, are inconsistent with the assumptions of the myopathic theory; such facts are: the way in which the disease will remain at a standstill in a muscle, the entire integrity of some bundles of muscles while the disease is making progress elsewhere, the immunity of muscles close to a diseased point, and the leaping over of the disease to distant muscles, etc.

The myopathic theory meets with a special difficulty in the frequent co-existence of an undoubted neurosis, progressive bulbar paralysis, with progressive muscular atrophy. In such cases the symptoms of the bulbar paralysis sometimes appear first, but more commonly are developed after the muscular atrophy has commenced. Friedreich thinks that in the former case there has been no true progressive muscular atrophy, but a paralysis with secondary atrophy, caused by progression of the myelitic process from the medulla oblongata to the fibres of the pyramids and the motor organs of the spinal cord (antero-lateral columns). But the clinical phenomena and the express statements of those authors who have observed the combination in question (Trousseau, Stein, Leyden, and others) are not very favorable to this attempted interpretation. In the latter case (as in the observations of Duménil, Duchenne, Valentiner, Wilks, Leyden, and others), Friedreich says that the muscular atrophy is to the bulbar paralysis as cause to effect. He regards as the mediate cause a neuritis, which ascends from the affected muscle along the nerve-tracks to the cervical part of the cord, extends into the medulla oblongata, and finally reaches the fibres of origin of the motor nerves of the head, which lie together on the floor of the fourth ventricle. But we have already seen how doubtful this chronic ascending neuritis is. No great importance can be attached to the circumstance that the upper extremities are chiefly or exclusively attacked in all this class of progressive muscular hypertrophies, for this is simply a general rule.

Far simpler and more natural is the theory established by Charcot and accepted by Kussmaul, which refers the frequent combination of progressive muscular atrophy and progressive bulbar paralysis to the genetic relationship of the two diseases from the neuropathic point of view. Both are due to analogous chronic changes (irritative atrophy) of motor ganglion-cells. In progressive bulbar paralysis these changes affect the groups of motor cells lying in the floor of the fourth ventricle, while in progressive muscular atrophy the cells of the anterior cornua are attacked, gradually and successively, so that many of them remain unharmed in the worst spots until the latest period of the disease. The latter circumstance also explains the fact that the trophic disturbances seldom affect all the primitive bundles of a muscle at once, and that the muscle still reacts to voluntary or electrical stimulation, even when its bulk is greatly lessened. The degenerative process itself, as Kussmaul states, has no specific character. We find both the ordinary gray degeneration and the chronic indurative myelitis, red softening, Clarke's "granular disintegration," or isolated pigmentary degeneration of cell-elements. All these have in common only the loss of the large ganglion-cells, which takes place in the successive manner above insisted upon. The distinction between progressive muscular atrophy and bulbar paralysis must therefore be sought only in the *difference of location of the original seat of the disease;* the cause of their combination, only in the *extension of the primary process.*

Among the separate symptoms of progressive muscular atrophy, the affection of the muscle itself needs no further explanation, as the material necessary for its comprehension, either from the myopathic or the neuropathic point of view, is contained in the preceding sections. The explanation of the collateral symptoms will naturally differ, according as it is drawn from the myopathic or the neuropathic theory; but many of them will be seen to admit but a forced and unsatisfactory explanation from the myopathic point of view. This, for example, is plainly the case with the disturbances of sensibility, which belong to the beginning or the course of the disease. They are scarcely compatible with the assumption of a primary chronic myositis, but may be explained naturally by a primary affection of the spinal cord, which, as the examinations show, not seldom extends to the entire gray substance, and in some cases to the posterior columns and the posterior root-fibres. The fact that common sensation is usually more affected than that of touch, and in some cases (Mosler and Landois) is exclusively affected, agrees entirely with a supposed original disease of the gray substance, and the increased reflex excitability which is observed in a few cases may be most naturally referred to the same.

Local differences of temperature can, at any rate, be regarded as a result of the myositic process, which first leads to a rise of temperature, and later, when the atrophy is developed, to a depression of temperature. It is harder to explain many local disturbances of circulation (cyanosis, arterial spasm), and the frequently associated atrophy of the integuments, and the arthropathy. These are more easily approached by the neuropathic theory, as they, after the analogy of other diseases of the spinal marrow, may be referred to vaso-motor-trophic disturbances of innervation, of spinal origin.

The oculo-pupillary symptoms, of course, depend either upon disease of the cervical sympathetic or of the anterior roots, or of the cervical spinal cord in the neighborhood of the last two cervical and the upper dorsal nerves. Voisin concludes from his case (before described), in which vaso-motor disturbances, elevation of temperature, etc., were absent, that the sympathetic cannot be the starting-point of those symptoms, but the anterior roots, since section of the latter, according to Bernard, produces contraction of the pupil and flattening of the cornea. Nevertheless, Bernard's statement that the fibres of the vascular nerves do not run in the anterior roots, but all originate from ganglia of the sympathetic, is not in agreement with the statements of other observers (Schiff). Besides, we often see only oculo-pupillary and no vaso-motor-thermic symptoms in cases of unquestionable lesion of the cervical sympathetic. To enter further into these questions is not now suitable, as the symptom in question is a very rare one.

Among the general symptoms, the fever which occurs in a few cases may be probably derived from the polymyositis. The hyperidrosis, which is a special concomitant of later stages, and is sometimes partial and unilateral, sometimes universal, is a symptom which may without doubt be of central or sympathetic origin (compare "Basedow's Disease"); but perhaps Friedreich's explanation is also admissible, namely, that there is a collateral fluxion to the cutaneous vessels, caused by obliteration of a large number of the finer muscular vessels in the rapid atrophy of the muscles. The hyperidrosis should therefore be interpreted like the sweats of trichina disease and tetanus, which are equally due to collateral arterial hyperæmia of the skin, dependent upon the impeded muscular circulation.

Diagnosis, Prognosis, and Treatment.

The diagnosis may present difficulties in the initial stage of the disease. It is especially possible to confound the disease with: *a.* genuine muscular atrophy, caused by direct, mechanical, traumatic agencies, as the atrophy of the interossei from pressure; *b.* secondary muscular atrophies in neuritis, which are likewise generally due to mechanical traumata or to rheumatism. To one or the other of these categories it is certain that many cases belong which are noted in literature as progressive muscular atrophy, and are said to have originated in traumatic or rheumatic injuries, etc. If the disease at a later period remains confined to the muscles originally affected, or to the region of a single nerve-trunk, progressive muscular atrophy can be excluded; we have nevertheless already given cases in which extensive atrophies, in the form of "myopathia propagata," have developed from an originally circumscribed seat of disease. (Cf. "Etiology.") I have also spoken there of the doubt whether these forms can be counted as muscular atrophy in its stricter sense. It is hardly possible to confound the disease with central or peripheral paralysis or paresis (though often accompanied by secondary atrophy), if we pay attention to the history of the case, its origin, the suddenness or gradualness of the appearance of the disturbance of function, the simultaneous or successive attack of the muscles, etc. Certainly there are also forms of paralysis of central origin, in which a gradual diffusion of the primary morbid process, or its appearance in multiple localities, may cause numerous muscles to be successively attacked ; but these very forms are easy to distinguish, as they are in general followed by no emaciation of the muscle, or at most by a tardy and slight wasting. In doubtful cases the symptoms of myositic irritation (fibrillary twitchings, etc.), and the results of electric examination, and above all, the microscopical examination of fragments of muscle which have been extracted, may lead to a conclusion. Of the mistakes arising from secondary lipomatosis of atrophied muscles we have already spoken under "Symptomatology."

The *prognosis* of progressive muscular atrophy is generally unfavorable, but it is by no means so absolutely hopeless as was supposed when the disease was first known, and as is still supposed by many. The possibility of saving at least a part of the muscles attacked cannot be denied as long as there exist in it uninjured or slightly injured muscular elements—as long, therefore, as *voluntary motility and the electrical reaction are not completely lost.* The volume of the muscle is, however, not a means of estimating the condition, since, as we have seen, its volume may be increased even when the proper muscular substance is wholly destroyed. The prognosis as regards life is deducible from the remarks upon the general progress of the disease. Cases are to be considered especially unfavorable which begin in a multiple form and with a tendency to a rapid course ; the prognosis is decidedly less favorable when the disease co-exists with a distinct congenital diathesis, or is accompanied by constitutional neuropathies, or preceded by bulbar paralysis, etc. Finally, those cases are less promising in which the disease takes its origin in the muscles of the shoulder or thorax, or passes rapidly to them, because this complication may affect to a dangerous extent the accessory muscles of respiration.

The treatment of the disease has many successes to boast ; but to gain them it is necessary to *begin as early as possible,* and to *persevere with untiring patience as long as possible!* Both the public and the physician often sin against both these precepts. I have repeatedly had the good fortune to discover the disease in a very early stage, when it was just attacking the interossei; but, failing to convince the patients of the gravity of their trouble, and to persuade them to adopt an energetic treatment, I have seen the disease develop to an incurable extent after months and years. Every specialist in this department has probably had similar experiences. Unfortunately, I cannot deny that the attending physician, through indolence or mistake, does not always show the degree of energy and perseverance in the treatment which is so especially desirable in this complaint. While in so many other cases we are obliged to resist the tendency to multiply useless remedies, in this case the policy of *laisser aller* is always to be rejected.

The treatment may be in part prophylactic; for in cases of inherited tendency, congenital predisposition, etc., the subject should be strengthened by a vigorous course of life, especially by a rational and regulated course of gymnastics, and should be prevented, if possible, from selecting a harmful profession, from excessive exertions, from rheumatic attacks, etc. When the disease has once appeared it may sometimes be a pressing necessity to give up the profession adopted, especially if injurious, and to change to one less harmful. It is true that we shall but rarely succeed in getting our advice adopted; it is just as in cases of co-ordinative professional diseases (writer's cramp and the like); there are usually external difficulties, and often still greater internal ones, which prevent the change being made; and yet there are a few successes obtained with intelligent and willing patients, which must console us for our numerous failures to persuade.

Absolutely nothing is to be expected of internal remedies; it is necessary to give a formal warning against them, lest the patient (as too easily happens) neglect the really important article of treatment under the belief that he is "doing something" for himself. In addition to the tonics (quinine, iron), especial use has been made of nitrate of silver, based on its occasional success in locomotor ataxia; also of arsenic, iodide of potassium, strychnia, and so forth. In the case of some patients who obstinately refused an electrical and gymnastic treatment, I have been able to use strychnia for a long time, both internally and in the form of subcutaneous injections; but in spite of long perseverance and large doses, which produced slight symptoms of intoxication, I never saw any effect whatever from the use of that remedy.

The only suitable and really trustworthy remedies are *electricity* and *medical gymnastics*. The latter is perhaps too much neglected, since the introduction into practice of the constant current and the great advances made in electro-therapeutics have raised the latter to the reputation of being almost a sovereign remedy in many diseases of the nervous and muscular apparatus. Yet undoubted successes have followed the use of *suitably localized gymnastics*[1] in this disease, and it is easy to see, from the theoretic point of view, that we possess in these active and passive motions an agent of especial efficacy in respect to the interstitial changes within the muscle. Its employment, of course, never constitutes a reason for not using electricity at the same time. Formerly electricity was applied only in the form of *induced current*, by the local use of which Duchenne obtained favorable results. But of late the *constant current* has gained reputation and deservedly, since there are cases in which the faradic excitability of the muscles is completely lost, while the galvanic may be in some slight degree retained, and since tolerable success has been achieved, even in this class of cases, by persevering galvanization.

Opinions are widely at variance in regard to the most suitable way of applying the constant current; they are, of course, much influenced by the prevalent theories of the disease. Remak, in accordance with the views already stated, recommended the so-called galvanization of the sympathetics, which has been observed to be followed by good results in the hands of Benedikt, M. Meyer, Guthzeit, Nesemann, Erb (in Friedreich), and others. Of especial interest is the case of advanced progressive muscular atrophy, described in full by Nesemann, in which galvanization of the sympathetic produced a complete cure. In this case, however, a relapse is said to have occurred, which did not yield to the previous method of treatment. M. Rosenthal observed no benefit from galvanization of the sympathetic; neither have

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[1] Cf. *M. Eulenburg*, Deutsche Klinik. 1856. Nos. 11–14. In a case which has recently fallen under my observation the process of *massage*, performed in Holland, was said to have brought the disease to a standstill.

I ever observed any special benefit from its use, and for a considerable time have confined myself in treating this disease to peripheral galvanization and faradization, which has given me results at least as satisfactory as when I have combined central applications with them. As regards details, I will here only state that local galvanic treatment, especially in old cases and when muscular excitability is very low, often requires at the outset extremely strong currents and methods of application (interruption, reversal, etc.); but, as the excitability increases, weaker currents may gradually be employed.

Regarding the *balneo-therapeutic* treatment, we have as yet no observations to be trusted. Scarcely anything could *à priori* be expected from the usual four weeks' courses in water-cures, in such a chronic and severe complaint; and the considerable number of patients who, with or without their physicians' orders, make journeys to the warm baths, cool baths, or sulphur baths, can only furnish a subject of regret to those who know that their time and money are simply wasted.

PSEUDO-HYPERTROPHY OF THE MUSCLES.

(Pseudo-hypertrophia musculorum.)

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The disease denominated Pseudo-hypertrophy of the muscles is clinically characterized by *an abnormal increase of size in certain muscles, accompanied by a diminution or loss of their functional energy, the direct cause of which is a chronic disturbance in the nutrition of such muscles* (*new formation of connective and fatty tissue, atrophy of the proper muscular elements*). The disease is certainly very closely related to progressive muscular atrophy, and is perhaps only a modification of the latter, due to peculiar circumstances.

History.

Of the cases belonging to this class, the first are those of Coste and Gioja (1838), Meryon (1852), and Rinecker (1860); but the diagnosis from the descriptions given is not at all certain. The credit of having been the first to state the specific quality of the disease, and to point out distinctly the contrast between the weakened function of the muscle and its excessive size, belongs unquestionably to Duchenne, who in 1861 published such a case under the name of "paraplégie hypertrophique de l'enfance de cause cérébrale." This was soon followed by publications from Kaulich (from Jaksch's clinic), Spielmann (from Schutzenberger's clinic), Stoffella, and Griesinger, the latter of whom first submitted excised portions of muscle to microscopic tests (applied by Billroth). The first complete report of an autopsy was published by M. Eulenburg and Cohnheim in 1866; the result in regard to the nervous system was negative; a monograph, summing up what was then known, was published soon after by Seidel. Since then the material has been increased by the addition of a number of cases, which, excluding doubtful ones, may amount to 110, while the clinical knowledge of the disease has been greatly promoted, and the nature of the pathologico-anatomical process lying at the base of it has been much discussed. But the latter point, as regards the muscle, is still *sub judice;* as for the question of implication of the nervous apparatus, either primary or secondary, there does not exist at present sufficient material for an exact discussion of it, and we are compelled to take refuge in simple hypotheses and possibilities. In the last few years progress has been made in two respects: on the one hand, more and more stress is laid on the connection between pseudo-hypertrophy and progressive muscular atrophy; and on the other, attempts are making to distinguish the disease in question from one far rarer, which has been incorrectly classed with it, namely, true hypertrophy of muscle (L. Auerbach, Berger, Friedreich). We shall speak of the latter disease in a special chapter.

Synonyms.—Recent as is our knowledge of the disease, and scanty as is the literature, the number of names proposed for it is nevertheless large. This is a suitable place to enter complaint against an ill fashion which is growing into repute, that every author who publishes a single case of disease thinks himself bound to clap to it a new name, whereby he only adds a fresh and needless obstacle to a common understanding. No name can fully and exhaustively express the nature of a disease, more especially of one so obscure and so little explained; under such circumstances, the best designation is always the most neutral, the most indifferent, which simply refrains from premature judgment and avoids positive error. Such a designation, for example, is that of "pseudo-hypertrophy." The expression "muscular hypertrophy," used by Kaulich, Stoffella, Griesinger, and others, is unsuitable, as neglecting to take into account the distinction between true and false hypertrophy. Of the other designations we will mention the following: Paralysie musculaire pseudo-hypertrophique, or paralysie myosclérosique (Duchenne); paralysie avec surcharge graisseuse interstitielle (Fritz, Tuefferd); sclérose musculaire progressive (Jaccoud); lipomatosis musculorum luxurians progressiva (Heller); atrophia musculorum lipomatosa (Seidel); diffuse muscular lipomatosis, myopachynsis lipomatosa (Uhde). By way of criticism upon these and similar terms, it will suffice to say that the expression "paralysis" does not correspond with the clinical nature of the disease, while the expression "lipomatosis" involves a view of the histological changes in the muscle which is one-sided, though not incorrect.

Etiology.

In estimating the causal elements of the disease, our attention is very strongly called to the predisposing effects of age and sex. As in progressive muscular atrophy, by far the greatest number belong to the male sex; of 86 that are adequately reported, 70 occurred in males and only 16 in females, which constitutes a ratio of nearly 9 : 2. This predisposition of the male sex is shown still more strikingly by the circumstance that when several cases occur in a family the male members are sometimes the only ones affected. In regard to the age, there is an apparent difference from muscular atrophy, since pseudo-hypertrophy makes its appearance chiefly during childhood, and, as a rule, before the close of the tenth year. Among 80 cases, in which it was possible to establish the date of commencement with accuracy, it began in the period from the first to the fifth year 45 times; from the sixth to the tenth, 22 times; from the eleventh to the sixteenth, 8 times; five times the disease made its first appearance in later life, or at least was first observed in adult men and women (aged 26, 30, 40, 41, 43 years). As Friedreich says, the disease seems to develop later on an average in women than in men; in one of the cases observed by me in the female sex this was the fact, as the disease first appeared at a ripe age, but in three other cases it began in the eighth year.

The influence of *heredity* and *congenital tendency* is felt here just as in progressive muscular atrophy, in the appearance of the disease among numerous members of one family (Barsickow tells of twenty-four cases in two families), and especially when brothers and sisters are attacked successively at the same age. Cases of two children of the same parents being attacked are numerous; cases of three brothers are reported by Heller, Wagner, and Seidel; of three sisters, by Lutz and myself; of four brothers, by Meryon. It is interesting that the three brothers observed by Heller had two fathers; their mother and two sisters were healthy, but a brother of the mother seems to have suffered from a similar complaint. These facts, as in progressive muscular atrophy, permit us to assume a diathesis as furnishing a foundation for the disease, at least in many cases; but in both diseases we are forced to confess our ignorance of the ultimate nature of the diathesis, and especially to leave undecided whether it should be regarded as a pre-formed morbid disposition in the muscular apparatus, or in certain (central, myotrophic) sections of the nervous apparatus. In favor of a neuropathic diathesis, and of an assignment of the disease to the group of so-called constitutional neuropathies, the circumstance may be mentioned that in some cases (not to speak of the frequent cases where progressive muscular atrophy co-existed) certain neuropathies, as insanity (Duchenne, Benedikt, W. Mueller, Schlesinger), have coexisted, or have preceded the pseudo-hypertrophy. In a few (Duchenne, Benedikt), epileptiform convulsions are said to have preceded the appearance of the disease. In several other cases the patients (children) possessed a specially low degree of intelligence, or there were also anomalies in the external development of the skull, either hydrocephalic configuration (Coste and Gioja, Duchenne, Friedreich), or asymmetry (Roquette, Hoffmann). Of circumstances of a more accidental nature which may have been related to the development of the disease, atmospheric influences—such as frequent over-heating and chilling—and damp, cheerless dwellings, and poverty, are mentioned. In a few instances the patients had previously suffered with acute diseases, especially measles (Stoffella, Griesinger, Hoffmann), or protracted scrofulous affections (Wernich, Seidel); in one case the cause is said to have been a fall from bed in the fourth year of age.

Symptoms and Course.

The cardinal symptom is increase of volume with simultaneous impairment of function in certain voluntary muscles; as a rule it is the *muscles of the lower legs*, though often also those of the *thighs*, which exhibit the symptom, either exclusively, or at least first of all, and most prominently. While, therefore, ordinary progressive muscular atrophy is characterized by the upper extremities being first affected, and in these parts certain muscles of the hand or shoulder first of all, we see in pseudo-hypertrophy, especially if it occurs in childhood, the lower extremities first attacked, and in these certain muscles of the calf, as the gastrocnemius and soleus. The development of a considerable excess of volume does not usually occur until the symptoms of diminished functional energy have existed for quite a time. The latter are especially marked in standing and in locomotion. The children show an increasing uncertainty in walking, tire very quickly, fall easily, and cannot rise without help. By degrees it grows harder to stand without support, the gait becomes waddling, or the child in walking straddles, depresses the point of the toe and raises the inner edge of the foot. As the process extends to the thighs, sitting down and rising from the sitting posture become extremely difficult; in the former act the patient lets himself drop mechanically upon his seat, and in the latter he seeks to assist himself by bracing his hands firmly against his thighs, unless this manœuvre is rendered impossible by progressive muscular atrophy of the muscles of the arm, which often co-exists. If he is examined while lying on his back, the legs are usually found spread apart, especially at the knees, while the feet are nearer each other; the latter are in the position of pes varo-equinus, with the soles turned toward each other, the heels drawn up quite high, the knee and hip-joints flexed. Plantar or dorsal flexion of the foot is executed badly or not at all; the same is true of supination and pronation, while the extension and flexion of the toes are usually unimpaired. Of the movements accomplished by the muscles of the thigh, extension at the knee-joint is usually the first to suffer; adduction and inward rolling of the leg is also made difficult, while flexion at the knee is often unaffected. If the function of the ileo-psoas is impaired, the patient is no longer able to draw up the thigh to the body, or to raise himself from a dorsal decubitus to the sitting posture. When the impairment of function is very slow in its progress, so that the patient continues to walk and stand for a comparatively long time, peculiar changes in the carriage of the body are developed, namely, a considerable lordosis of the lumbar portion of the spine and a compensatory kyphotic curve in the dorsal portion.

This position is probably caused by the instinctive effort to obtain a surer support for balancing the trunk upon the lower extremities, by moving the centre of gravity forward, and thus preventing the body from falling forward. At a later period the kyphotic position may be very much exaggerated, owing to an implication of the muscles of the loins and back, especially the quadrati lumborum and sacro-lumbales; or, if the muscular affection is mainly limited to one side, a strongly marked scoliosis arises.

These progressive impairments contrast strongly with the outward appearance of the parts attacked. The leg and foot, and often the thigh also, look uncommonly large and voluminous, and are more or less enlarged in circumference. In cases where the process begins upon one side or remains unilateral, it is easy to ascertain the increase by measurement; but it is often sufficient to compare the patient's leg with that of a well child of the same age, and in many cases a brief period of observation suffices to demonstrate a considerable growth, especially in the calf. The parts hypertrophied, when felt by the hand, usually give the impression of a spongy, hard-soft mass of fat, a great lipoma, without a trace of muscular tension and resistance. In advanced cases this is true both of the calves and of the muscular masses of the front and outside of the leg, and the front and inside of the thigh; sometimes also of the posterior pelvic and dorsal masses. It can often be demonstrated that the spongy, lipoma-like feel depends greatly upon an increase of the subcutaneous fatty tissue, and is therefore most noticeable in places where nature has provided an abundant panniculus. On the other hand, single muscles often exhibit a strikingly firm and hard consistency, although they may be more or less deficient in functional power, and even completely insensitive to the electrical stimulus or that of the will. These differences in consistency are dependent on the fact that the disease is sometimes limited to excessive lipomatosis, while in other cases this is combined with extreme proliferation of interstitial connective tissue and retraction of the newly formed tissue. (Compare "Anatomy of the Disease.")

When the disease ascends and finally attacks the upper extremities, it seldom does so in its original form; the form of ordinary progressive muscular atrophy is much more frequent, and we may observe a combination of the latter, in the arm, with a pseudo-hypertrophy of the muscles of the trunk and lower limbs (or of the legs below the knees only). This is especially the case with children, but may also occur in adults, as is seen in a case observed by me (a cook aged forty-four), in which there was pseudo-hypertrophy of one lower extremity, with great atrophy of the upper extremities. On the other hand, there is a disease which does occur in adults, and which deserves the rank of a special form of disease, under the title of "hypertrophia musculorum progressiva adultorum" (Benedikt), in which the arms, especially the shoulders, are the regions first attacked, with hyper-volumen of the muscles affected. But these cases, for reasons later to be stated, must probably be counted in with the form of disease designated as "true muscular hypertrophy." The process in pseudo-hypertrophy is not such that an increase of volume can be detected at one given time in all of the muscles of the lower limbs and trunk which are diseased (*i. e.*, functionally weakened), for muscles which are extremely hyper-voluminous may lie side by side with other muscles which, while equally feeble, are yet lax, flaccid, and reduced in volume. The cause of this difference can only be explained by anatomical explorations. We can only speak here of our previous remarks upon the secondary and *compensatory* lipomatosis of the atrophied muscles which accompanies progressive muscular atrophy. Finally, it remains to be said that the invasion and progress of the disease is not always symmetrical, and that, even after the disease has lasted a long time, the hyper-voluminous muscles of one side may correspond to muscles of normal or even less than normal volume on the other side. In the above related case of pseudo-hypertrophy and progressive atrophy in an old lady, I saw a preponderant affection of the left lower and the right upper extremity. I have observed a similar crossing in several other cases.

As in progressive muscular atrophy, the muscles affected are not infrequently subject to *fibrillary twitchings;* only they are less striking to the eye, and, on account of the masses of fat which cover them, are less easily seen than in the atrophied muscles of the arm. The *mechanical excitability* of the muscles is variously increased in a few cases.

The *electrical reaction* of the affected muscles is influenced by two important facts: first, the atrophy of the proper muscular elements; and secondly, the lipomatosis of the muscle (and, to some extent, of the integument lying over it). The consequence of both these facts must naturally be a weakening of the reaction, the former producing a diminution of the contractile mass, the latter involving a great increase in the resistance to the passage of the electric current. It is therefore evident that as the disease makes progress the faradic and galvanic contractility of the muscle must continually diminish. This diminution may go to the extent of completely abolishing the faradic and galvanic reactions, as I have observed in the above case in all the muscles of the left leg and foot, as well as in the trunks of the nerves (peroneus and tibialis).[1] In the muscles of the right leg, which were much less altered in function and volume, the faradic and galvanic reaction under direct application was not lost, but considerably diminished.

The excitability and conductivity of the nerve-trunks can at least remain quite unaltered for a long time, and, since in this case one of the factors of weakened reaction (increased resistance, from interposition of fatty masses) is absent, a sufficiently strong current may thus indirectly produce muscular contraction as long as contractile fibres exist. Yet by degrees the nerve-trunks also exhibit a falling away in excitability, especially for the induced current, which is probably due to secondary degeneration in the peripheral nerves (see below). It is certainly hard to reconcile with anatomical facts the statements of the entirely normal electrical condition which certain authors make (especially Schlesinger, in a case lately observed in Meynert's Clinic). Among the qualitative changes in galvanic reaction, the prolongation of the contraction at closure (closure-tonus) and the

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[1] I cannot tell where Friedreich found the expression he quotes from me ("integrity" of the electrical excitability) upon page 317 of his work, especially as he elsewhere quotes me (p. 283) as saying precisely the reverse.

"Lückenreaktion" of Benedikt deserve mention. The latter consists in a weakening or disappearance of the anodic opening reaction as the current is increased in strength, and its reappearance when the current is made still stronger. It is probably due (according to Fick) to the twofold action of the current, whereby the excitability of a nerve is increased, while its conductivity, on the other hand, is diminished, thus causing a break ("Lücke") in the series of reactions, at the point where the increased effect of the current upon excitability is neutralized by the increased resistance to conductivity.

Electro-muscular sensibility has been found increased by several observers (including myself), and by some diminished. The calf-muscles, which are the oftenest affected, are well known to afford the best means of testing this very uncertain phenomenon, and I therefore am inclined to allow these observations somewhat more value than similar ones usually possess.

Disturbances of sensibility, of other kinds, are not infrequent at the beginning and during the course of this disease. Pains in the back and the small of the back, piercing pains in the lower extremities, are in many cases reported as premonitory symptoms. They seem sometimes to follow the course of certain nerves, the cruralis or sciatic (Rakowac), or are felt chiefly in the region of joints (the fold of the groin, the hollow of the knee, etc.); in a lying posture they become less or disappear entirely, while they are increased by attempts to flex the joints. Paræsthesiæ are also common, especially the feeling of cold and of formication, while anæsthesiæ, in any considerable degree, are exceptional.

Anomalies of *circulation*, of *temperature*, and *nutrition* in the parts affected must also be regarded as frequent. The diseased regions, especially the lower half of the leg and the foot, have, in the majority of cases, a peculiar coloration, being partly pale, partly bluish, with a mottled appearance, owing to the accumulation of dilated veins in certain spots; their temperature is decidedly lowered, at least in the advanced cases, sometimes standing at 8° or 9° R. lower than that in the axilla. The secretion is also lessened, as a rule; the skin is smooth and dry, somewhat disposed to crack, and seems, as it were, thinned by being stretched over the voluminous muscles. The condition of the subcutaneous tissue is especially striking. While in progressive muscular atrophy the integuments not rarely waste, and especially the panniculus adiposus, we see, on the contrary, in pseudo-hypertrophy, along with the excessive development of fat in the muscles, an increase of the subcutaneous fatty tissue which is sometimes very considerable.

The *course* of the disease is essentially determined by the extent to which the muscular affection has spread. In the majority of cases the great skeletal divisions bounded by the joints seem to present limits, which, however, are not at all impassable. Thus we often see the process confined to the muscles of the lower half of the leg; in other cases the muscles of the thigh participate at a later period; and still later, the muscles of the posterior surface of the pelvis and back. Pseudo-hypertrophy of the upper extremities is rare, as above stated, and always secondary; the increase of volume is mostly confined to a few muscles (deltoid, triceps), while the others are sometimes normal in size, sometimes diminished.

The process extends most rarely to the muscles of the head, the neck, and the front part of the trunk; yet there are a few cases showing that these muscles are not wholly free, in which a swelling of the muscles of chewing, also of the tongue (Coste and Gioja, Chvostek), the sterno-cleido-mastoids, the recti abdominis, etc., has been reported. Whether the hypertrophy of the heart, observed in a few cases (Coste and Gioja, Rinecker), can be brought into connection with the general muscular affection, may remain undetermined. The extreme of diffusion is, perhaps, that which was attained in a case reported by Duchenne, in which almost all the voluntary muscles (including those of the face), except the sacro-lumbales and pectorales, were hyper-voluminous. This was the case of a boy aged ten, whose intelligence was weak: it resulted fatally. (See "Anatomical Changes.") The nature of the disease is such that there are fewer circumstances leading to a fatal result than in progressive muscular atrophy; for the muscles of respiration, whether proper or accessory, are much less often affected. Yet the great danger which such an event involves is by no means out of the question, for pseudo-hypertrophy of the lower extremities is not rarely united, as we have seen, with progressive atrophy of the upper segments of the body. Whether children or adults who are attacked with pseudo-hypertrophy can attain to an advanced age is still questionable, as the cases hitherto reported give no sufficient account of the matter. In fatal cases death was commonly preceded by disease of the organs of respiration, in the form of acute affections of the larynx and trachea, pneumonia, bronchitis, or chronic phthisis of the lungs ; in one case (W. Mueller) by the symptoms of dementia paralytica, in one by scarlatina.

Anatomical Changes.

The pathological changes in the muscular tissue can be studied in the living subject by macroscopic and microscopic examination of portions of muscle extracted for the purpose. As regards the methods of exploration (excision, harpooning), the remarks made in connection with progressive muscular atrophy are entirely applicable. Excision is to be rejected ; harpooning only is allowed, but does not always lead to satisfactory and demonstrative results, especially when, as is often the case, the panniculus adiposus is thickened. For the present this procedure furnishes the chief material for studying the changes in the tissue of the diseased muscle. At present there exist but very few complete reports of autopsies, only four in all (Eulenburg-Cohnheim, W. Mueller, Barth, Charcot). Besides these there exist only the older statements of Meryon, very aphoristic in form, and in recent times a very fragmentary account by Martini. A somewhat larger number of examinations have been made upon the living by Griesinger-Billroth, Heller, Wernich, Duchenne, Russel, Knoll, Rakowac, Friedreich, Schlesinger, and myself.[1]

Different observers, unfortunately, differ widely in the statement of the anatomical conditions observed by them, and their disagreement is still wider when they come to interpret and to draw conclusions. It is impossible to enter upon special details in this place, and the reader must be referred to the literature of cases, and the pretty complete collection in Friedreich's work. Nothing further than a synopsis and grouping of the principal results can be attempted here.

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[1] Auerbach's and Berger's cases are not included here. (Cf. "True Muscular Hypertrophy.")

As seen by the naked eye, the fragments of muscle removed from the living subject, and the general mass of the affected muscles at autopsies, appear discolored, pale or yellowish white, and only in less advanced cases of a faint light-reddish tint. If the disease has made great progress, the muscles of the corpse have a soft, inelastic, almost doughy consistency, their cut surface exhibits an exquisite fatty lustre, and their whole appearance differs so little from that of the subcutaneous tissue that only the striped appearance here and there visible enables us to recognize the muscle as such; in places abundantly covered with fat (as the gluteal region) the boundary line between muscle and panniculus adiposus is sometimes hardly traceable. Such muscles as are attacked without being hypertrophied look like those described under the head of Progressive Atrophy.

The condition most frequently shown by the microscopic examination is an abundant development of fat, interstitial or even interfibrillary, which must be regarded as the cause of the increase of volume in the affected muscle. It is, however, very probable that the development of fat is not the primary fact, but that it is preceded by a stage of hyperplastic development of connective tissue, so that the whole must be spoken of as an irritative, inflammatory process, as in the case of progressive muscular atrophy. The proliferation of connective tissue springs first from the perimysium internum, and from the adventitia of the small vessels; the newly formed connective tissue appears to be especially rich in small-celled (sometimes spindle-shaped) elements and nuclei, which undergo secondary transformation into fat-cells. At any rate, during the early stages of the process, it is common to see the fat-cells arranged in rows like a rosary in the midst of a striped basal tissue, which is explained, it is true, by some authors (Cohnheim) as the empty, collapsed sarcolemma tubes of the vanished muscular elements, but is decidedly affirmed by others (Friedreich) to be hyperplastic interfibrillary connective tissue. The assumption that the formation of fat is preceded by a hyperplastic proliferation of connective tissue has been already stated by Billroth (in Griesinger's first case), and is shared by Wernich, Knoll, Duchenne, Jaccoud, and others; upon this rests the designation of the disease as *paralysie myosclérosique* or *sclérose musculaire progressive.* This assumption is supported by the fact that in some cases the process seems to have been arrested at the stage of formation of connective tissue, or at least the transformation into fatty tissue seems to have taken place to but a slight extent (Russel, Knoll, Rakowac).

If the interstitial fat is more abundant, coarser bundles of muscular fibre are first pressed apart, then the single primitive fibres are separated in the same way, so that at last an entire muscle seems uniformly permeated by fatty tissue. The primitive fibres become attenuated at the same time, and at last they disappear altogether, leaving behind them only the collapsed empty tubes. This condition I have myself observed in a fully developed pseudo-hypertrophy of the calf-muscles, with complete immobility and want of response to the electric stimulus. But where the disturbance of function is slighter the cross stripes of the primitive fibres can always be found, though the fibres for the most part are very much thinned in respect to diameter. There is one striking phenomenon, to which Cohnheim first called attention, and which has been confirmed by Knoll, Barth, W. Mueller, and myself, namely, the occurrence of decidedly hypertrophied fibres along with others which were more or less attenuated. These hypertrophied fibres, of twice or thrice the usual thickness, generally present quite a normal appearance, and have only in spots somewhat of a finely granulated look; Barth found their transverse stripes very indistinct. Cohnheim and Knoll have further found a dichotomous or trichotomous division of the hypertrophied elements—a condition resembling that which Friedreich demonstrated in a case of progressive muscular atrophy.

That the muscular elements themselves may be implicated in the irritative process, is further confirmed by the multiplication of the muscular nuclei (Friedreich, Charcot), and by the occasional fine granular cloudiness of the muscular fibres, which disappears after the addition of acetic acid, and must therefore be looked upon as a parenchymatous exudation. In W. Mueller's case muscular fibres with granular contents were seen, presenting a varicose appearance with alternate enlargements and constrictions.

As a final stage of the process we may mention, besides the simple atrophy of muscular elements, a fibrillary fissuration (Barth, Charcot, Friedreich) or waxy degeneration (Charcot, Friedreich), while proper fatty degeneration seems to occur only by way of exception and in a few fibres.

The post-mortem appearances observed by Martini are quite unique and at present seem hard to interpret, and so are those which Schlesinger obtained by excision from the living body. The former states that in the transversely-striped substance of the primitive bundles there occurs a peculiar formation of fissural spaces, which gradually enlarge and fill with a sero-albuminous fluid. With the progressive disappearance of the striped substance, these fissures run together, and at last form tube-shaped fibres, which in section are ring-shaped, or (if they retain their septa) appear as disks perforated like a sieve, with a number of round openings. Martini therefore designates this process as "tabular" or "serous" atrophy of muscle. In Schlesinger's case there was found an abundance of fibrillary and undulated (wellenförmiges) tissue, among which were muscular fibres in various states: some retaining their cross stripes, some in the condition of granule-heaps of a distinct yellowish color, some irregular in outline, some in the form of roundish or longish masses; destruction of muscular masses by granular degeneration. Neither proliferation of the interstitial tissue nor increase in the volume of individual muscular fibres could be observed, but a considerable vascularity was everywhere present. Schlesinger believes himself justified in assuming upon the ground of this observation that the abundant fibrillary tissue did not originate by the ordinary process from connective tissue cells, but from muscular fibres; that the disease therefore appeared first in the muscular fibres themselves (as a process of chronic inflammation), and that the interstitial tissues were therefore drawn to participate in the process at a later time. Probably this case is related to those of Rakowac, Russel and Knoll, above described.

As regards the changes in the nervous system, we are at present confined to the few results of autopsies already mentioned.[1] In the case of Eulenburg-Cohnheim the result of a naked-eye examination was entirely negative. The brain, cord, and their envelopes, also all the nerve-roots and the trunk of the sympathetic, presented no abnormal conditions. The sciatic and crural nerves of both sides were somewhat flattened, but showed no other changes. In the case examined by W. Mueller, which was, however, complicated with dementia paralytica (in a woman aged thirty-four), there was found an extended degeneration in the cord, especially in the lateral columns, consisting in a thickening of the interstitial connective tissue with an increased number of cells, a diminution of the diameter of some of the primitive nerve-fibres, with granular change of the primitive sheaths and partial disappearance of the axis-cylinders. In the gray substance the ganglion-cells were uninjured as far as to the middle of the lumbar swelling; farther downwards there was an *attenuation of the anterior cornua, especially on the right, with atrophy of the greatest part of the ganglion-cells contained in them;* the intervening substance was unusually dense and poor in axis-cylinders. The trunks of peripheral nerves (peroneus, tibialis, sciatic) exhibited a considerable increase in the volume of their interstitial connective tissue, with groups of fat-cells interposed here and there; a similar development of connective tissue was also found in the anterior roots, which were colored gray, but without any injury to the primitive fibres.

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[1] The oldest observations by Meryon (1852), the relation of which to the disease in question is certainly somewhat doubtful, remarked no anomalies in the nervous system.

Barth likewise found extensive changes in the cord and the peripheral nerves, but is inclined to interpret them as secondary. They consisted of wedge-shaped or roundish spots, distinctly gelatinous, irregularly distributed in the white substance of the anterior and lateral columns of the cord. In these spots the nerve-fibres were few; their place was taken by a finely granular substance, traversed by large, full blood-vessels, and containing numerous corpora amylacea. In the anterior cornua there was found only a very small number of ganglion-cells, and the vessels were much dilated. The sciatic nerves were strikingly broad, and the several bundles were pressed asunder by a deposit of fat. The sympathetic was not examined.—Martini mentions the lipomatosis of the nerve-trunks only incidentally; his promised publication of the condition of the cord has not yet appeared.—In the case examined by Charcot (the boy of ten years observed by Duchenne) the examination of the cervical enlargement and the upper dorsal part of the cord gave an absolutely negative result, and the same in respect to the anterior cornua and the large ganglion-cells contained within them. The anterior roots and the nerve-trunks (sciatic, median, radial) were likewise normal. When the muscles were examined, entirely normal portions of nerve were often found, and only once (in the psoas) was there in them a very marked hypertrophy of the axis-cylinders.

Theory of the Disease and Analysis of the Symptoms.

The important question to be answered is, whether the pseudo-hypertrophy of the muscles should be looked upon as an independent form of disease, or only as a special development of progressive muscular atrophy. If the independence of the disease is granted, the question as to its primary myopathic or neuropathic origin will have to be considered. If the second alternative is adopted, the same question will have to be put, but (according to the statements made under progressive muscular atrophy) without any immediate hope of definite decision.

The chief arguments in favor of the essential identity of progressive muscular atrophy and of pseudo-hypertrophy are as follows: (1.) Agreement in the fundamental histological changes of the muscle. Although upon this point, especially in respect to pseudo-hypertrophy, we have seen that there are many unsolved contradictions, yet we may be tolerably sure of as much as this, that in both diseases the first stage consists of a chronic irritative process, proceeding from the interstitial connective tissue and affecting secondarily the muscular elements—*a chronic myositis accompanied by interstitial hyperplasia of the connective tissue* (Friedreich). In the subsequent course, in the complications of this originally identical process, differences certainly arise which require special explanation. Another and a weighty argument is (2.) the extremely frequent coincidence of pseudo-hypertrophy of single muscles with diminution in the size of others, near or remote, which are similarly impaired in their functions. Sometimes, and not rarely, a marked pseudo-hypertrophy in the lower extremities will be combined with the usual form of progressive muscular atrophy of the upper part of the body. Finally (3.), essential agreement in respect to etiology and clinical course may be noted. Points held in common are: the preponderance of the male sex; the influence of a predisposing diathesis, usually congenital, often referable to hereditary tendency; the occurrence of several cases in one family, the successive attack of several brothers and sisters at a certain period of life. It is plain that the two diseases may take the place the one of the other, as the example communicated by Russel shows, where in one family two brothers were attacked by progressive atrophy, and a third by pseudo-hypertrophy. As regards the clinical coincidence, I will only mention the numerous common points in the functional and electrical condition of the muscle, in the manner of extension of the muscular affection, in the later consequences (secondary deformities), etc.

If, on the other hand, we group together the *principal points of difference*, we find them relating first to the manner in which the histological process further develops itself in the diseased muscles. But this difference is only partial. The secondary implication of the muscular tissue, the wasting and final disappearance of the muscular elements, partly by simple atrophy, partly in the form of fibrillary fission or waxy degeneration, is common to both processes, on the whole. It might be said, at most, that a participation of the muscular fibres in the proper irritative processes seems to be somewhat more frequent and marked in pseudo-hypertrophy than in progressive atrophy; but we find in the pseudo-hypertrophic muscles, in addition to and subsequent to the initial hypertrophy of connective tissue, an excessive development of fatty tissue, which in a later stage transforms the whole muscle and its neighborhood into a spongy mass of fat, and at the same time often makes the muscle enormously voluminous. (In a few cases, as the observations of Russel, Knoll, and others, prove, it seems that the external increase in bulk may originate solely in interstitial proliferation of connective tissue). Nevertheless, even in this excessive development of fat in pseudo-hypertrophy, the distinction from progressive atrophy seems to rest solely upon quality. We have seen that the latter disease is not rarely accompanied by a secondary lipomatosis of the muscles attacked, and that the atrophied muscles are not seldom enabled by this "compensatory" process to increase in volume up to the point of equalling or even surpassing their former size. This secondary lipomatosis after atrophy especially affects those muscles which are the favorite seat of pseudo-hypertrophy. Friedreich very properly points out how much irritative processes in general favor or provoke the pathological development of fat, of which fact numerous instances are found in the history and etiology of lipomas and of other lipomatous proliferations.

The etiological and clinical differences between pseudo-hypertrophy and progressive muscular atrophy are most strongly expressed in the facts, that the former is almost peculiar to childhood, while the latter arises more frequently in adults in the middle period of life; that, further, hypertrophy begins (almost without an exception) in the lower extremities, and very often remains confined to them, while progressive muscular atrophy, as a rule, prefers the upper extremities and the muscles of the trunk. And yet these distinctions, far from widening the gulf between the diseases, perhaps help to fill it; perhaps they point to the cause which gives rise to a divergence in two cases originally based upon one and the same histological process. In childhood, as has been stated in connection with progressive muscular atrophy, there exists a circumstance especially favoring the development of the disease in the lower extremities, namely, the great efforts made with these limbs in the acts of standing and walking. If the diathesis be present we should not be surprised to find just these parts first attacked; and might not childhood present conditions favoring and accounting for the occurrence of the process in that form which characterizes pseudo-hypertrophy, namely, the excessive development of fat in the muscles attacked? In fact, such conditions seem really to exist. Friedreich calls attention to the fact that the fattening of cattle is favored by youth, by fat-producing nutriment, and by rest, and that all these circumstances meet in the pseudo-hypertrophy of childhood. The fact that the lower extremities usually suffer condemns the child at an early period to continued inactivity, and his nourishment consists, often to a very great extent, especially in the lower classes, of the fat-forming cereals. It is true that the pseudo-hypertrophic development of fat is not a case of simple passive fattening, but of an accessory lipomatosis supervening on an actively inflammatory process; yet it is plainly shown by the frequent considerable increase of the panniculus adiposus and the lipomatosis of the nerve trunks (Barth, Martini) that there exists a certain tendency to general adiposity. Putting these facts together (though all difficulties be by no means solved), we may affirm that Friedreich's view is very probable, which considers pseudo-hypertrophy to be only "*a form of progressive muscular atrophy modified by the peculiar strength of the diathesis and by certain special attributes of childhood.*"

Whether the muscular affection is to be regarded as primary, or as a secondary affection dependent on a previous lesion of trophic nerve-centres, must therefore remain undecided, especially since the pathologico-anatomical material for determining the intensity and extension of the lesions of the nervous system is at present very scanty. The same fact interferes with the interpretation of the accessory symptoms. Those disturbances of the sensibility which are often present, especially the pains and paræsthesiæ which follow the course of certain nerve-trunks, may perhaps be referred to interstitial proliferation of the connective tissue of the nerve trunks (compare the case reported as above by W. Mueller), and to the consequent irritation of the sensitive fibres.—The anomalies of circulation and temperature in the affected parts (the bluish, mottled look, the dilatation of the veins of the skin, the diminution of temperature, and so on) may depend on the loss of muscular action, and the consequent interference with the return of the venous current, or perhaps they may also be due to collateral diversion of circulation to the skin in consequence of the obliteration of numerous muscular blood-vessels (Friedreich). But the later factor may be of subordinate moment, as is shown by the circumstance that some observers have found the affected muscles universally rich in blood-vessels (Schlesinger), while the skin was affected as described.

Diagnosis, Prognosis, and Treatment.

The *diagnosis* of pseudo-hypertrophy cannot be made with certainty in its early stages, when the increase in the size of the muscle is not evident, or is very slight; but a close observation of its etiology, and the application of functional and electric tests, can help us at least to form a very probable opinion. The surest evidence is furnished, of course, by a microscopic examination of portions of muscle extracted by the harpoon, provided the necessary precautions be observed—plunging the harpoon as deeply as possible, on account of the frequent thickening of the panniculus—and that the operation be repeated. In later stages of the disease no greater mistake is likely to happen than that of diagnosticating true muscular hypertrophy, but the functional and electric tests are sufficient to guard against this.

The *prognosis*, as has already been intimated in speaking of the course of the disease, is somewhat more favorable in regard to life than that of the common form of progressive muscular atrophy, unless perhaps there exists a tendency to ally itself with the latter disease. But, as regards the disease, the prognosis is still less favorable. Those muscles in which a considerable amount of accessory fat-formation has occurred seem to be less susceptible of a functional restitution than those in which the chronic process of inflammation is unaccompanied with formation of fat; and for this reason a cure is but rarely to be looked for in the advanced stages of the process. Statements to the contrary, made by a few authors, may often be based upon a mistaking of this disease for true muscular hypertrophy.

The *treatment* must in general be conducted according to the same principles as in progressive muscular atrophy. A careful prophylaxis is of special importance; children congenitally predisposed, or the younger brothers or sisters of persons who have already become affected, must be shielded as far as possible from injurious atmospheric influences, too early and exhausting locomotion, excess of fat-forming nourishment, etc. When the disease has commenced, such internal remedies as tonics, quinia, iron, cod-liver oil, etc., are as useless as in progressive atrophy; the same seems to be true of iodide of potassium, although its use is defensible upon the rational ground of its known power to promote the resorption and disappearance of fatty tissue. As regards the use of localized gymnastics, we have as yet too few observations; but it seems to promise some advantage in the initial stages of the disease, though at a later period no success is to be expected, at least as regards the restoration of the affected muscles. Massage and hydro-therapeutics seem to have been of value in some cases.—By far the greater part of the cases have been treated by electricity for a longer or shorter period, sometimes with the induced, sometimes with the constant current. Duchenne in particular has seen favorable results from faradization (*i. e.*, induced current), and in two cases has even observed a cure (!) after treatment for several months. The constant current has been chiefly used in galvanization of the sympathetic. While Benedikt obtained considerable benefit from this procedure, other authors (Erb, Chvostek) observed no essential gain, or even (Roquette) an apparent loss. The local method, of applying faradization and galvanization to the muscles affected, would seem therefore to deserve the greater confidence.—Consecutive deformities require a special orthopedic treatment, which, however, owing to the continuance of the original disease, can only palliate the trouble. In a case contributed by Uhde, where an advanced degree of pes varo-equinus existed, the deformity was removed, and the power of walking somewhat improved, by tenotomy of the tendo Achillis (which was followed by an abscess in the calf).

TRUE MUSCULAR HYPERTROPHY.

(Hypertrophia musculorum vera.)

L. Auerbach, Ein Fall von wahrer Muskelhypertrophie. Virchow's Archiv. Band 53. S. 234 and 397.—*O. Berger*, Deutsches Archiv für klinische Medicin. 1872. Band IX. S. 363.—*Benedikt*, Wiener med. Presse. 1872. Nr. 9. Compare also his Elektrotherapie, 1869. S. 186.—*Friedreich*, Ueber progressive Muskelatrophie, über wahre und falsche Muskelhypertrophie. Berlin, 1873.

True muscular hypertrophy is clinically characterized by *a noticeable increase in the volume of the muscles, while the muscular force may be increased, or may remain normal, or may even be slightly diminished.* Its anatomical basis consists of an *actual hypertrophy of the muscular elements.*

Partial hypertrophy of the striped muscles, especially of the tongue, has very long been known, as has also the existence of muscular hypertrophy in connection with general hypertrophy of one-half of the body, of one extremity, or one side of the head. But the observation of pure cases of muscular hypertrophy in the extremities, and the separation of these from pseudo-hypertrophy, with which they were confounded, or were considered as the initial stage, is one of the most recent discoveries. The number of observed cases is, therefore, extremely limited. It is doubtful whether an observation of Graves's belongs to this class, in which sciatic pains were associated with a hypertrophy of the leg. The first certain case is that of L. Auerbach, in which the diagnosis could be confirmed by an examination of excised pieces of muscle. We must also regard as sufficiently well established cases two reported by Berger (and perhaps also a third case by the same), several by Benedikt, and one by Friedreich.

In regard to causation, it is to be remarked that four cases (Auerbach, Berger) occurred in soldiers who had performed severe forced marches during campaigns; two of Berger's cases were preceded by ileo-typhus or gastric fever, and a third by a gunshot injury of the thigh, without lesion of the larger vessels and nerves. Benedikt's and Friedreich's cases were in adults; in that of Friedreich the subject was a maid-servant thirty-three years old, but in this instance the disease was probably congenital; at least, the left arm was said to have been larger than the right at birth. (Benedikt's proposed designation of "hypertrophia musculorum progressiva adultorum" seems, therefore, not improper upon the whole.)

The cases hitherto observed have mostly affected the upper extremity (Auerbach, Benedikt, Friedreich), and, as a rule, only *one* upper extremity has been attacked; in Auerbach's and one of Benedikt's the right, in Friedreich's the left. In those cases also in which the disease was seated in the lower extremities (Berger), it was always confined to one of them. In the cases of Auerbach and Friedreich the increase of volume affected almost all the muscles of the arm, and especially those of the upper arm and the region of the shoulder. The circumference of the upper arm was, in Auerbach's case, 5½ and 6½ cm. greater, and that of the forearm 5 cm. greater than the corresponding measures upon the unaffected side; similar differences are given by Friedreich, as also a considerable increase in the breadth of the hand (2½ cm.), caused by hypertrophy of the interossei and enlargement of the ball of the little finger. In Benedikt's cases the hypertrophy was essentially limited to the muscles of the shoulder (deltoid, pectoralis major, teres major and minor, serratus ant. magnus). In Berger's cases—which possess, in many points, the usual characteristics of pseudo-hypertrophy—the muscles of the calf were the first and the most severely affected.

As regards the functional energy of the diseased muscles, Auerbach found that a greater display of force could be made for brief exertions, but that fatigue occurred more quickly than is usual. Berger and Benedikt found the power somewhat lessened; Friedreich found it at least as great as upon the healthy side. The consistency of the muscles attacked was hard and firm, and on contraction they bellied out strongly and compactly. Fibrillary contractions are mentioned by Berger only. The mechanical excitability was found increased by Benedikt; under galvanic tests he found the "Lückenreaction" and closure-clonus. Auerbach found the excitability under constant currents equal on both sides, but weakened for induced currents on the diseased side. Erb (in Friedreich's case) found it normal on both sides for both classes of currents, with the exception of the ball of the thumb, which looked as if atrophied, and exhibited a corresponding diminution of excitability in the flexor pollicis brevis and opponens, both under direct and indirect applications of electricity. In Berger's cases, on the other hand, the excitability under both classes of current was diminished, and so was the reflex excitability.

Of the condition of extracted pieces of muscle, reports have been made by Auerbach, Berger, and Friedreich. Those of Auerbach are especially careful. The pieces of the deltoid and biceps which he cut out exhibited a perfectly normal structure, but an enormous breadth of the muscular cylinders; their diameter in the deltoid averaged 120 μ, while specimens taken by way of comparison from dead bodies measured only from 58 to 75 μ. The muscular cylinders of the diseased (right) biceps had diameters reaching to 165 μ, those of the left only 110 μ. By measurement of the breadth of the separate striæ Auerbach proves that the enormous increase in breadth cannot be due solely to the contraction following exsection (rigor mortis), but depends on actual hypertrophy. A comparative count of the muscular nuclei in one cubic millimetre of normal and one of hypertrophied muscular substance showed but slight differences, so that a considerable growth of nuclei in the hypertrophied muscle must also be assumed. The investigations both of Berger and of Friedreich likewise demonstrated a considerable increase in the thickness of the muscular fibres, amounting even to a doubling, while the cross striations were well retained. Symptoms of growth of interstitial fat or connective tissue were entirely absent, both here and in Auerbach's case.

Among the muscular symptoms presented by the above cases, *disturbances of sensibility and circulation* deserve especial mention. The former were found only in Berger's cases (violent neuralgiform pains, formication, anæsthesia); in the other cases the sensibility of the skin was normal. In Auerbach's case a livid coloration of the diseased arm is mentioned. Benedikt found in one case symptoms which entitled him to assume an *affection of the sympathetic;* the face was redder and the secretion of sweat was increased upon the affected (right) side; the pupil was dilated and the sympathetic was sensitive to pressure. In another case there existed extensive vaso-motor paralysis in the face, the neck, on the breast, and over the shoulder-blades, which disappeared after galvanization of the sympathetic.

In regard to the *theory of the disease* the first question to settle is, whether the muscular hypertrophy should be considered an independent disease, or only as a first stage of pseudo-hypertrophy. The latter view is taken by Berger and Auerbach. Auerbach assumes that the primary excess of nutrition is followed later by a process of retrograde metamorphosis with atrophy of the muscular bundles, and a filling in of the enlarged interstices with connective tissue and fat. The appearance of this stage of retrogression is hastened by the abnormal conditions of circulation, especially by the disproportionate growth of the capillaries enveloping the muscular cylinders, which transfer their nutritious materials in less abundance than formerly to the interior of the muscular cylinders, and more abundantly to the interstitial substance. In this way hyperplasia of the latter and atrophy of the muscle-cylinders would necessarily be produced. But, ingenious as is this theory, the transition from true to false hypertrophy (or the super-induction of interstitial growth of fat and connective tissue) has not been demonstrated by a single positive observation, while the favorable clinical results and the permanent functional and electrical integrity of the diseased muscles, etc., are entirely inconsistent with such a view. The question whether the true muscular hypertrophy should be regarded as a genuine muscular lesion or as a tropho-neurosis (Berger) can hardly be discussed at present, owing to the entire want of material for its decision. The affection of the cervical sympathetic observed in Benedikt's case may probably be regarded as simply a case of combined disease, such as is observed in some instances in progressive muscular atrophy.

Among the symptoms, one deserves especial attention, namely, the readiness with which fatigue of the muscle occurs (Auerbach), or even a general weakening of its power (Berger, Benedikt). Auerbach finds the cause of this singular phenomenon in the fact that the muscular blood-vessels do not increase in proportion to the growth of the muscular cylinders, and that the restoration of the muscular force after expenditure must take place too slowly; perhaps also the intra-muscular terminations of the nerves are not developed in sufficient proportion to conduct the volitional impulses readily. Benedikt, too, attributes the loss of function chiefly to an inadequate growth of the nervous elements; also to pressure of the swollen muscular substance upon the terminal nervous apparatus, and to a probable primary lesion of the central trophic ganglion-cell system. Friedreich, on the contrary, in explaining the rapidity of exhaustion, admits but one hypothesis, that of disproportion between the muscular and the vascular masses. The increased power for brief exertion, as in Auerbach's case, rather gives reason to suppose (according to Friedreich) that the hypertrophy of the muscle is accompanied *pari passu* by a hypertrophy of the terminal motor nerve-disks, or of the components of the motor nerve-fibres, which serve in the conduction of the volitional impulse—that is, the primitive fibrillæ composing the axis-cylinders.

EPILEPSY AND ECLAMPSIA.

NOTHNAGEL.

EPILEPSY AND ECLAMPSIA.

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Epilepsy.

The general literature cited above may be consulted, and besides: *E. Loebenstein-Loebel*, Wesen und Heilung der Epilepsie. Leipzig, 1818—*John G. Mansford*, Investigations into the nature and causes of epilepsy.—*Portal*, Natur u. Behandlung der Epilepsie. Uebers. von Hille. Leips. 1828.—*Herpin*, Prognose und Behandlung der Epilepsie. Deutsch bearb. von Frank. Quedlinburg and Leipzig, 1854.—*Delasiauve*, Traité de l'Épilepsie; Histoire, Traitement, Médecine légale. Paris, 1854.—*Schroeder van der Kolk*, On the minute structure and functions of the spinal cord and medulla oblongata, and on the proximate cause and rational treatment of epilepsy; translated by W. D. Moore. London, New Syd. Soc. 1859.—*A. Kussmaul* and *A. Tenner*, On the nature and origin of epileptiform convulsions caused by profuse bleeding, and also of those of true epilepsy; translated by E. Bronner, London. New Syd. Soc. 1859.—*Brown-Séquard*, The various works of this author on epilepsy are cited in a foot-note to the text.—*Sieveking*, On epilepsy and epileptiform seizures, etc. 2d ed. London, 1861.—*Ch. Bland Radcliffe*, Epilepsy and other convulsive affections. London, 1859.—*Althaus*, On epilepsy, etc. London, 1866.—*Russell Reynolds*, Epilepsy: its symptoms, treatment, and relations to other chronic convulsive diseases. London, 1861.—*Gonzalez Echeverria*, On epilepsy; anatomo-pathological and clinical notes. New York, 1870.—*Nothnagel*, Ueber den epileptischen Anfall. Volkmann's Sammlung klinischer Vorträge. Leipzig, 1872.—Additional short monographs and the essays on epilepsy in the journals are cited in the text.

Introduction.

The history of the development of the doctrine of epilepsy affords one of the most brilliant examples of the important aid which experimentation on animals may furnish towards furthering our knowledge of a diseased condition. This disease was known as far back as medical literature extends into antiquity. And this is not surprising, for the violent form of its coarsest manifestations could not escape even the most superficial observer. In the Hippocratic writings we find it characteristically described. In course of time a few additions were made to the symptomatology, and some new remedies—one generally as little to be relied on as another—were brought into use. Beyond this point, however, the knowledge of epilepsy made no essential progress for many centuries. Naturally there was no lack of attempts to explain its symptoms and its nature. Innumerable hypotheses, clever and foolish, anatomical and physiological, philosophical, even theological, were advanced with this object in view. None of them raised the veil a hair's breadth; none contributed anything whatever to *positive* knowledge. The last twenty years alone have advanced us at one time more than the previous twenty centuries taken together. The experimental researches of two investigators furnished the starting-point, although some isolated but unsatisfactory attempts had already been made in this direction. In a series of experiments, which were carried out in a masterly manner, fit to serve as a model, Kussmaul opened the way to an understanding of the *epileptic seizure.* Brown-Séquard, by numerous experiments upon animals, first indicated a way, by following which it is perhaps possible to attain a more accurate knowledge of the *epileptic state.* In the course of recent years the efforts of various other investigators have been added to these two fundamental works, and their results will be alluded to in the proper place.

Until a short time ago, in spite of the number and variety of the names for it which have been gradually adopted,[1] no scientific doubt existed that epilepsy was a perfectly definite disease with well-marked symptoms. As Boerhaave defines it, Epilepsia est abolitio subita omnium functionum animalium, cum augmento motuum vitalium et motu convulsivo in omnibus musculis corporis, so it has always been esteemed as a distinct disease, to the definition of which the idea of "chronic" has only very lately been added.

During the last ten years a marked reaction has set in against this view. In the first place one group of symptoms after another, which from a clinical point of view apparently showed the features of epilepsy, has gradually been detached in proportion as our increasing knowledge proved that definite anatomical changes in the brain or other organs were the direct causes of epileptic attacks. This is true of uræmic seizures; also of all those epileptiform convulsions in which demonstrable gross alterations exist in the cranial cavity, the effect of which taken altogether may be comprised in this, that either directly or by simple mechanical conditions they occasion an anæmia of the brain substance. In all these cases, and quite properly, we no longer speak of epilepsy, but only of epileptiform spasms, as symptoms of the conditions in question. In this category belong the epileptiform, or as they are also called in this case, "eclamptic," seizures in profuse hemorrhages, in hydrocephaloid, in great hyperæmias, in all intracranial diseased conditions which are accompanied by considerable diminution of space, and particularly if they develop rapidly, as in the case of important hemorrhages. In our opinion, however, this use of the term "eclamptic" is confusing and unjustifiable. (Compare the section on Eclampsia, and also the sections in the twelfth volume of this work on Anæmia and Hyperæmia of the Brain.)

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[1] We give some of the Latin designations, without alluding to the numerous popular names in various modern languages: Morbus sacer, major, herculeus, comitialis, convivalis, mensalis, insputatus, viridellus, vitriolatus, sonticus, caducus, lunaticus, fœdus, sideratus, scelestus, dæmonicus, deificus, astralis, St. Valentis, and St. Joannis, Analepsia, Apoplexia parva, Passio Caduca, Perditio, etc. Compare Joseph Frank, Die Nervenkrankheiten. 4th vol.—Attempts at an explanation of several of these names may be found in Josat, Recherches historiques sur l'Épilepsie. Paris, 1856.

There can be no doubt that such a pathology as that just sketched agrees with the present position of science. In this way a definite group of cases, which were formerly described as "symptomatic," is done away with; the spasmodic seizures have rather been relegated to the rank of a symptom merely, the anatomical basis of which is well known.

For the same reason the so-called "toxæmic" epilepsy, as a distinct disease, has been allowed to fall into disuse. The epileptic manifestations, which may occur in various acute as well as chronic forms of poisoning, and among which lead-poisoning has always played a prominent part, are called forth either directly or indirectly by the action of the poison upon the central nervous system, in a way which cannot here be more particularly discussed. In these cases, too, the epilepsy is only a symptom, and represents no special form of disease.[1] This whole group, therefore, of the "symptomatic and toxæmic" epilepsies will be left out of consideration in the following account; the individual cases find their place with the corresponding primary diseases.

In very recent times, however, we have even gone a step further. For quite a long time another form, "sympathetic or reflex epilepsy," was recognized in addition to those mentioned. Under this name were comprehended those cases in which an irritation of any kind, affecting any sensitive nerve whatever could be proved, by the effects of treatment, or could at least be assumed, to be the source and starting-point of the disease. If the irritation and consequently the state of spasm were persistent, as, *e. g.*, in consequence of a cicatrix, we called it epilepsy; if it were of an acute kind, *e. g.*, from the eruption of a tooth in a child, we called it eclampsia.

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[1] This is also true of the epilepsy from drinking absinthe, on which Magnan especially has recently laid so much stress.

Of late even this mode of treating the subject has been rejected by several investigators as erroneous and confusing. Russell Reynolds especially, whose views on the pathology of this disease are highly esteemed, confidently maintains the opinion that the so-called sympathetic or reflex epilepsy is to be relegated to the group of "convulsions" simply, and must be completely separated from epilepsy, with which it has nothing in common save the symptoms of the peculiar convulsions: "epilepsy should be regarded as an *idiopathic* disease, *i. e.*, a morbus *per se*, which is to be distinguished from eccentric convulsions, from toxæmic spasms, from the convulsions occurring in connection with organic changes of the cerebro-spinal centre, and from every other known and recognizable disease." (R. Reynolds, l. c., p. 33.) Reynolds allows, to be sure, that an irritation in the foot, in the mucous membrane of the digestive tract, in the lobes of the brain, etc., may result not only in simple convulsions, but occasionally even in "epilepsy;" in spite of this, however, he maintains that a sharp distinction should be established. In the case of convulsions "a condition of increased irritability in the reflex centre" merely has been reached; in epilepsy, on the other hand, this condition has "acquired *an existence of its own*, and the exalted irritability thenceforth depends upon an altered nutrition, which continues even after removal of the original eccentric source of irritation." In their external manifestations, in their duration through a period of years, the two conditions may be wholly alike. The simple convulsions, however, are distinguished by their pronounced and constant relation to demonstrable irritations, and by yielding to the appropriate treatment directed to this irritation.

From the weight of Reynolds' authority we feel obliged to enter somewhat more into detail with regard to his views, though still but briefly. We cannot deem them correct, and we regard the division which he makes as not fortunate. It cannot be doubted that in epilepsy a peculiar condition, an alteration *per se*, exists in the central nervous system. But this "epileptic change,"[1] to the further discussion of which we shall return below, is, in our opinion, also found in many cases that Reynolds calls eccentric convulsions, and can be explained simply according to Pflueger's laws of reflex action. The only two grounds which he adduces for the division do not render it necessary. For at any rate no constant relation can be proved to exist between the single seizures of eccentric convulsions and any irritation whatever. It is a well-established fact that in persons who have become subject to epileptiform seizures as a result of a cicatrix or some such cause, these seizures do not occur only or exclusively after irritations of the cicatrix, but also quite spontaneously or after mental changes. The former, however, would necessarily be the case if we had to do with "reflex manifestations" only; for certainly, from a physiological point of view, the conception of these has no other significance than that the same connecting link of irritation always precedes them; but if convulsions appear without being preceded by a definite external irritation, then a pathological change of some other kind must already have taken place in the reflex centre. In such cases, therefore, we shall be obliged to assume a peculiar change in the pons and medulla oblongata. But even the fact that recoveries take place after removal of the irritation is by no means proof of an essential difference, as Reynolds represents it, for it chiefly concerns cases which come under treatment soon, in which, therefore, the epileptic change has not yet become inveterate; and it is well known, too, that "idiopathic" epilepsies are relatively the most curable if they are still recent. If the "reflex epilepsy" have been present for years, it does not always disappear at once on the removal of the peripheral irritation, but single paroxysms make their appearance, proving that we have to deal with something more than a simple chain of reflex manifestations, for otherwise there would be nothing to excite any further paroxysm after the removal of the definite pathological irritant.[1] Finally, it does not favor the idea of simple reflex convulsions that (except in the case of wounds of the head) the first paroxysm follows only a considerable time, perhaps a week, after the establishment of the peripheral irritation.

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[1] In the course of this account we shall always employ this expression and not that of "epileptic condition," which was formerly used for it, so as to render impossible any confusion with the "état épileptique," which is employed in French in a wholly different sense.

Accordingly, the form of reflex epilepsy ought to be retained. Only this name does not appear to us comprehensive enough. We shall subsequently have to explain that the epileptic change is excited and brought about not only by the peripheral nerves, but also, though less often, by the spinal cord, and in less frequent cases by the brain. If, therefore, those cases are designated as *idiopathic* or primary epilepsy in which no such excitation can be proved, it is perhaps not injudicious to characterize by a name which commits us to no theory, viz., "*secondary*," all those other cases in which an affection of the brain, the spinal cord, or the peripheral nerves, is present as a source of excitement and a starting-point for the disease. Meanwhile, we are agreed with Reynolds in excluding from epilepsy any epileptiform seizures which occur quickly for some few times in consequence of an external irritation, and disappear just as quickly again after its removal. The teething convulsions of children furnish the type of these. *Functionally*, of course, the process is here the same as in true epilepsy; but in those parts of the brain which will be mentioned further on, no evidence can be found of that more permanent change which must necessarily be present for the characterization of epilepsy. The name of *Eclampsia* may well be employed for these conditions, which Peter Frank very properly designated as acute epilepsy. We shall enter into this more fully when speaking of *Eclampsia*.

Thus, on the one side the domain of the epileptic conditions has been more closely limited, in part correctly and in part wrongly, as we have just described, while in another direction it has within the last ten or twenty years attained a markedly increased extent. A whole series of cases, the clinical picture of which would formerly have fitted into the frame of no one of the recognized groups of symptoms, are to-day reckoned as epilepsy, following the example of Trousseau and especially Griesinger. The latter writer preferred the designation of "*epileptoid conditions*," but we do not hesitate to call them simply epilepsy. While formerly, in characterizing the disease, emphasis was laid upon the convulsive symptoms, it is true that now, from consideration of the "little attacks" (*petit mal*), the opinion has gradually gained ground that the loss of consciousness is to be regarded not only as of equal significance, but in it is to be sought the controlling element of the epileptic attack. The study of the epileptoid conditions has forced this way of looking at it more and more into prominence. In fact, the varying forms under which they present themselves, and which will be described further on, lead to the conviction that not a complete abolition of consciousness even, however short it may have been, and without any muscular spasm, is necessary to characterise the disease as epilepsy, but that a simple dizziness is sufficient,—in fact, any alteration whatever of the mental activity occurring paroxysmally, such as hallucinations and the like. A common causal connection, however, viz., an altered, *i. e.*, increased or diminished, degree of fulness of the vessels of the brain, embraces all these varying features of the paroxysms one with another, and makes them comparable with the great attacks of the ordinary forms of epilepsy. Of this we shall say more hereafter.

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[1] A pronounced case of this kind has been recently reported by *Hitzig* in his "Untersuchungen über das Gehirn," p. 187.

It can be proved from the course of the epileptoid conditions that, although characterized sometimes by the most peculiar symptoms, they agree essentially with the ordinary forms of the disease, and that the *epileptic change* lies at the root of them too. For instance, they are often preceded by a distinct indubitable injury, or during their progress spasmodic symptoms occur, at first of limited extent, but which gradually develop the well-known characteristic features of the disease; or again a major attack may appear suddenly, when for a long time there had existed only the indefinite epileptoid attacks. Recently, however, Westphal[1] has insisted quite properly that we should not go too far in this direction, that we should not at once declare every patient to be an epileptic who occasionally presents epileptoid or epileptiform accesses. According to his experience, he regards these attacks as among the most frequent symptoms in mental and nervous diseases of the most varied nature, without the character and essential nature of these diseases being determined by their occurrence in single instances, *i. e.*, without their being thus stamped as epilepsy proper. We shall endeavor further on to determine where the boundary lines should be drawn in this matter, what patients with epileptoid seizures may yet be regarded as suffering from epilepsy proper.

While, therefore, we consider the establishment of these conditions in true epilepsy as essentially correct in the present state of our knowledge, we cannot say the same for "*epil. spinalis*," a term which has been brought into use especially by Brown-Séquard. Of course, if we use this designation for those cases in which an actually existing epilepsy is developed in consequence of an affection of the spine, it would have a certain justification. Still it is superfluous, for here the name of secondary epilepsy, as above proposed, is in our judgment amply sufficient. We must, however, very decidedly protest against the abuse which has recently come into vogue of describing as spinal epilepsy the clonic and tonic spasmodic seizures which occur as a symptom in spinal affections, which remain confined to the extremities or even to the legs, and are not accompanied by any trace of mental changes. With just as much propriety could we speak of a spinal accessory or median epilepsy in the case of clonic twitchings of the muscles of the fingers or neck which proceed from a peripheral affection of the median or spinal accessory nerve. In our opinion it is most judicious to let the expression fall entirely into disuse, for on one hand it is unnecessary, and, on the other, it only leads to confusion.

From all that has been said so far, it follows plainly that it is not possible at the present time to give a brief definition of epilepsy. In the following propositions, the reasons for which are in part to be found in the course of the account, are comprehended the most important characteristics of the disease.

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[1] Ueber Agoraphobie, Archiv f. Psychiatrie u. Nervenkrankheiten. Vol. III. 1872.

Up to the present time epilepsy is only a clinico-symptomatological conception, such, *e. g.*, as apoplexy, but not at all an anatomical one, as *e. g.*, cerebral hemorrhage. Nevertheless, it must, according to all our present knowledge, be adhered to as the designation of a perfectly definite morbid condition.

This morbid condition is composed of two factors: of a persistent change in the central nervous system on the one hand, and on the other, of external symptoms, which occur partly in paroxysms, and are in part continuously present. The mere form of the paroxysms does not alone warrant the assumption of an epilepsy, for they may be produced in a precisely similar way by various other causes.

The anatomical changes which underlie the group of symptoms called epilepsy, and which, in a given case, are somewhat variable and more or less clearly marked, are to be sought for in those sections of the central nervous system which include the pons, the medulla oblongata, and the upper part of the cervical medulla. We are still unaware whether these changes, which constitute the essence of epilepsy, also take place still further up or down in the brain and spinal cord, but this is not probable. We must, of course, disregard such changes in the brain and cord as are a consequence of the seizures.

These changes and processes in the above mentioned portions of the brain are up to the present time unknown in their essence and nature, and either come into existence there primarily or are excited secondarily, either by the peripheral nerves, the cerebrum, or the spinal cord.

The group of symptoms in the individual seizures corresponds physiologically to an excitation, or again to a fatigue of all or of the single motor and vaso-motor nerves, the nuclei, *i. e.*, the reflex centres of which, are situated in the parts of the brain referred to.

Finally, the idea of chronicity belongs to the characterization of epilepsy. In the so-called acute epilepsy or eclampsia the central changes, which properly constitute the essence of the disease, are wanting.

Experimental Researches.

It seems proper to give before the clinical exposition a brief account of the series of experiments which have been undertaken for the purpose of gaining a clearer insight into the nature of epilepsy. In bringing them together in this place, our chief object is to render the subsequent explanation of the pathology of the disease easier to understand, and to enable the reader to obtain a connected survey of the material which can be utilized in building up a pathology of the affection. The experiments may naturally be divided into two series: the one refers to the mode of origin and the character of the epileptic change, the other to the genesis and nature of the seizures.

Brown-Séquard[1] for some twenty-five years has occupied himself almost uninterruptedly with the artificial production of epilepsy in guinea-pigs, a species of animals especially well fitted for the purpose. In these animals, after the wounding of various portions of the nervous system, an epileptic state is gradually developed; as after lesions of the spinal cord, section of one or both of the sciatic nerves,[2] wounding of the medulla oblongata, or, finally, of the pedunculus cerebri, or the corpora quadrigemina. Of course we cannot in this place repeat all the multifarious details, and therefore present only those which are most important. With the peripheral nerves this effect follows after section of the internal popliteal, the trunk of the sciatic, and the posterior roots of the nerves of the leg; with the spinal cord it most readily follows after complete section of one lateral half, but also if the posterior columns, the gray posterior horns, and a portion of the lateral columns are simultaneously divided, or if only the two posterior, lateral, or anterior columns are separately cut through, or if the whole spinal cord is divided, or finally, if a simple puncture is made in it. These wounds produce the most decided effect when made in the tract from the eighth dorsal to the second lumbar vertebra, though the result sometimes follows even after a wound of any other portion. After a short time a state of increased excitability is developed; spasmodic twitchings in certain groups of muscles first appear, and then complete epileptic seizures. On an average, and most frequently, from four to six weeks (11–71 days) elapse before their first occurrence. The attacks, in part, break out spontaneously, or they are determined by the irritation of a definite portion of the skin, which Brown-Séquard designates the *epileptogenous zone.* This includes the cheek and the antero-lateral region of the neck (trigeminus and occipitalis), and is always found on the same side upon which the cord or sciatic nerve was wounded, but, on the contrary, is on the opposite side in wounding of the crus cerebri. This zone is indicated by a certain degree of anæsthesia. Slight irritations of it, pulling of the hairs, and the like, are sufficient to produce the attack. Sometimes the epileptogenous zone extends downwards almost the whole length of the vertebral column. The attacks now occur with varying frequency, sometimes very often. After the epileptic state has lasted for a long time, perhaps for years, it gradually subsides, and in the same proportion the anæsthesia of the epileptogenous zone diminishes. If it has been produced by a section of the sciatic and a rapid union of the ends of the nerve has taken place, the epilepsy also disappears again rapidly. Finally, it is also a very noteworthy fact that the young of such guinea-pigs may be spontaneously epileptic, *i. e.*, without any injury having been inflicted on them.

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[1] His publications on this subject are scattered through various journals; the results of his earlier labors are contained in his book: "Researches on Epilepsy; its artificial production in animals, and its etiology, nature, and treatment." Boston, 1857. His later investigations are found chiefly in the Journal de physiologie de l'homme, Vols. I. and III., 1858 and 1860, and in Vols. I. to IV. of the Archives de Physiologie normale et pathologique (*Brown-Séquard*, *Charcot*, *Vulpian*), 1868-1872.

[2] These facts had already been observed by *Vulpian*; compare Arch. de physiologie, etc., 1869, p. 297; and Analoges bei Rana temporaria, by *Schiff*.

This is what is most essential in Brown-Séquard's very numerous experiments. The same facts have been determined by Schiff and Westphal. I may add from my own experiments upon guinea-pigs that in a very strong animal, in which the cord, with the exception of the posterior columns, was divided at the level of the third lumbar vertebra, the most violent seizures set in at the end of only thirty-six hours, death occurring in the course of them from œdema of the lungs; and Schiff saw them set in after three and four days. Moreover, the experiments have been confirmed in regard to cats and rabbits, although not as yet in a thoroughly satisfactory manner. Schiff[1] has also observed convulsions in dogs at the end of the first week after wounding the cord in the cervical region, yet the animals at the same time retained consciousness, quite contrary to the case of Brown-Séquard's guinea-pigs.

Westphal[2] produced an epileptic state likewise in guinea-pigs in another way. If the animals are given light blows upon the head, an attack of general convulsions results. Then, for a considerable time nothing special is to be noted ; but after the lapse of some weeks the animals behave exactly in the same way as those in which the spinal cord or the sciatic has been wounded, *i. e.*, upon irritation of the above-mentioned epileptogenous zone, or even apparently spontaneously, general convulsions break out in which coma is also sometimes present, but at other times appears to be absent. This state lasts for from six weeks to six months. At the autopsy Westphal constantly found small hemorrhages in the spinal cord and in the upper part of the cervical medulla.

Hitzig[3] has recently furnished a further interesting contribution to the pathogenesis of epilepsy, as he has been able to cause it by the artificial production of morbid processes in the cortical portion of the brain. If, *e. g.*, he removed the cortical centre which controls the anterior extremity, well-marked epileptic seizures, beginning spontaneously, set in after a longer or shorter time, once on the very next day, or after from three to six weeks.

The significance of these various series of inquiries as to the etiology and pathology of the epileptic state in man we shall have numerous occasions to allude to in the further course of this account.

Experiments have advanced us decidedly further in the knowledge of the epileptic seizure. A. Cooper[4] even sought to prove by experiments, which were, to be sure, very meagre and defective, that ligation, equivalent to compression of the carotids and vertebral arteries, induced epileptic seizures. Travers, likewise, and Marshall Hall insisted upon the resemblance of eclamptic and epileptic paroxysms to the symptoms occurring after the loss of great quantities of blood. Kussmaul, however, was the first, in a work carried out in conjunction with Tenner, to arrive at a great series of important and well-established results. We can, of course, in this place only quote the most essential of them. In the first place, Kussmaul showed incontrovertibly that, when the brain is rapidly deprived of arterial blood, either by bleeding or by the ligation or compression of the four great arteries going to the head, in vigorous animals not etherized, coma and general epileptic twitchings are invariably produced by it, and that these symptoms quickly disappear when the obstacle to the current is removed, unless it have remained too long.

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[1] Lehrbuch der Physiologie. Lahr, 1858–59.

[2] Berliner klin. Wochenschr. 1871. No. 38.

[3] Untersuchungen über das Gehirn. Berlin, 1874. p. 271.

[4] Guy's Hospital Reports. Vol. I. London, 1836.

Direct examination of the brain, by means of the method introduced by Donders, proved the actual presence of anæmia of that organ. Further experiments taught him that the starting-point of the convulsions was to be sought in the excitable motor portions of the brain lying behind the optic thalami; that, moreover, the spinal cord during the seizure serves only as a conductor, but that anæmia of this part does not result in twitchings. A critical view of the symptoms present in man, and the comparison of them with the results of experiment, led Kussmaul and Tenner to the conclusion: 1st, that a circumscribed anatomical change in the brain cannot be regarded as the immediate cause of epileptic seizures (because the coma necessarily points to a simultaneous participation of the cerebrum); 2d, that the proximate cause of the seizures might not be any change of long continuance or constant, or even a grosser one appreciable by the anatomist, but merely a change of a transient kind. In order to prove the hypothesis that epileptic accidents may be induced by means of vascular contraction, through the medium of the vaso-motor nerves, they then endeavored to produce anæmia of the brain and the paroxysms by faradization of the cervical sympathetics. They succeeded in this, however, only in a single instance, and then only in an animal in which, besides this, both subclavians and one carotid were ligated. I, too, have attempted this latter mode of experimentation, with a negative result. The reason of this failure is clear: as I have proved,[1] only a portion of the nerves of the cerebral vessels run through the cervical sympathetic; according to A. Schultz,[2] indeed, only abnormally.

Further than this, I have endeavored (l. c.), by irritation of the peripheral sensory nerves, to produce, as a reflex effect, constriction of the arteries of the brain, and thus a cerebral anæmia. My positive results have been confirmed by some very successful experiments of Krauspe,[3] while avoiding several sources of error proved by Riegel and Jolly[4] to exist in my experiments; and certainly this was antecedently probable, judging from our former physiological knowledge.

It has therefore been proved by Kussmaul that cerebral anæmia stands in definite relations to the epileptic seizure; we shall have to enter into the details further on. How is the case now with hyperæmia of the brain? Kussmaul and Tenner tested this question by ligating the internal and external jugular veins in rabbits, after section of the sympathetics in the neck, but no epileptic tremblings broke out. Quite a number of years later, to be sure, Landois[5] maintained this view, though Hermann and Escher[6] re-established the correctness of the statements of Kussmaul and Tenner in regard to rabbits, notwithstanding that in the case of cats they were able, by occlusion of the veins which lead the blood from the brain, to produce exactly the same group of symptoms as in occlusion of the cerebral arteries, although it was somewhat more slowly developed.

Besides this, Kussmaul and Tenner made experiments to test a well-known hypothesis of M. Hall,[7] according to which the course of the symptoms in the epileptic seizure would be the following: 1st. Excitation of a sensory, spinal, or cerebral nerve, or even direct central excitation; 2d. Thereby are caused: *a*, a reflex tonic spasm of the muscles of the neck especially (trachelismus), by which the veins of the neck are compressed (sphagiasmus), and thus the comatose symptoms excited; *b*, a reflex tonic spasm of the muscles, closing the rima glottidis (laryngismus), leading to asphyxia, and through this to the general convulsions. Kussmaul and Tenner established the already well-known fact that occlusion of the larynx produces coma and convulsions. They were obliged, in accordance with the experiments adduced in the foregoing paragraph, to pronounce against the trachelismus theory. On the other hand, Russell Reynolds (l. c.) has shown that a contraction of the muscles of the neck produced by electrical irritation results in stasis in the veins of the neck and cerebral disturbances.

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[1] Virchow's Arch. Vol. XL.

[2] Petersburger med. Zeitschrift. Vol. XI.

[3] Virchow's Arch. Vol. LII.

[4] Ibid. Vol. LIX.

[5] Centralbl. für d. med. Wissensch. 1867.

[6] Pflueger's Arch. Vol. III.

[7] Memoirs on the nervous system. London, 1837. Also in various other places.

With regard to the anatomical starting-point of general convulsions in the epileptic seizure, numerous experimental studies have been published. As already related, Kussmaul and Tenner established it in the parts of the brain lying between the spinal cord and the crura cerebri, consequently in the pons and medulla oblongata; Brown-Séquard and Schiff arrived at analogous conclusions. Then I have shown[1] that the convulsion centre, *i. e.*, that circumscribed spot from which the whole body of the voluntary muscles may be thrown into tonic and clonic spasms through reflex excitation, is to be sought for in the pons. In reference to the spinal cord, Kussmaul and Tenner have already established the fact that in convulsions from excessive bleeding it serves as a conductor only; that no convulsive motions starting from it can be excited by anæmia. This fact I have been able to establish as indubitably correct, in opposition to certain recent statements, which, moreover, do not seem to be based on experiments.[2] From this, however, the conclusion must not be drawn that also in epilepsy proper the spinal cord acts simply as a conductor during the seizure; for it is of course well-known that, under certain circumstances, complete epileptic convulsions may take their origin from it alone, and this phenomenon has even led to the designation epilepsia spinalis.[1] Still, some interesting experiments of Brown-Séquard seem to favor the view that the function of the spinal cord in the seizure is only that of a conductor. In epileptic guinea-pigs he witnessed the absence of convulsions in the hind leg of the side on which the spinal cord was divided; but besides this he also found the following: if a guinea-pig has been made epileptic by section of one posterior column and the neighboring gray substance, while the anterior column is left untouched, the voluntary movements remain intact on both sides, and nevertheless the convulsions are absent on the side of the section. From this Brown-Séquard concludes that certain portions of the spinal cord convey the impulses to voluntary, and others those to the spasmodic movements.

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[1] Virchow's Archiv. Vol. XLIV.

[2] Ibid. Vol. XLIX.

Perhaps still another conclusion can be added to this last experiment. We mentioned above (p. 191) that it is wholly unknown whether the medulla spinalis partakes in the change which constitutes the essential feature of epilepsy. This experiment appears to us, at all events, not to favor that view.

Brown-Séquard then made the further observation on the epileptic animals that the commencing seizure, in which the head is regularly turned towards the side of the injury, may be arrested by a sudden vigorous turn towards the opposite side, and its further development prevented. Who does not, in this connection, at once think of the popular remedy of forcing open the thumb and the like? There will also be an arrest of the seizure if the epileptogenous zone is touched not lightly but strongly, if it is burned or cut; in fact, it thereby loses its epileptogenous capacity. The seizure may also be arrested if a strong stream of carbonic acid is brought to bear upon the mucous membrane of the throat. This last communication, which was only very recently made by Brown-Séquard, has, indeed, already been contested by Filehne,[2] who never succeeded in cutting short the seizure by ever so strong a stream of carbonic acid.

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[1] Compare especially *Hallopeau*, Des accidents convulsifs dans les maladies de la moelle épinière. Paris, 1871.

[2] Archiv von *Reichert* und *Dubois*, 1873.

Etiology.

If the causes of epilepsy are to be studied, a correct method of examination must of course be followed. This would require no special mention, if a false way of drawing a conclusion were not often made use of so soon as we have to do with a special case, in deciding from any fact whether it be the cause of the epilepsy or not. We will give an example. Some one, who has hitherto appeared well, is violently alarmed, experiences an epileptic attack, and afterwards remains epileptic; therefore, it is decided, the fright was the cause of the epilepsy. We consider this wrong. The only direct sequence furnished by the facts is that the fright provoked the epileptic seizure.

In our opinion it cannot be kept too prominently before us, even in regard to the etiology, that we must draw a clear distinction between the central epileptic change, in the sense in which we have presented the idea above, and the symptomatic expression of it, viz., the seizures. The question as to the causes of epilepsy, then, breaks up into two parts:

1. *What influences produce the epileptic change?* and
2. *What produce the outbreak of the symptoms?*

There can scarcely be room to doubt that a change such as that which lies at the root of epilepsy and constitutes its essential character, a change of so thoroughly chronic a kind, can also, in analogy with other morbid processes, develop only slowly and gradually. It is of course conceivable that some condition, having an acute and transient action, shall, on some one occasion, give the first impulse to the development of the change, just as occasionally the development of a malignant tumor may be caused by an injury; but these cases form the marked exceptions.

If we analyze the individual cases of epilepsy according to the various conditions, which, with varying frequency to be sure, may, in accordance with our experience, lead to the development of the epileptic change, they naturally fall into three great classes:

a. Cases in which the disease is produced by factors of a kind not appreciable anatomically, which at times affect the nervous system alone, at others the whole organism.

b. Cases in which lesions of a definite anatomical kind affecting the nervous system act as the cause.

c. Cases in which no causes are conceivable, or can be assigned with any degree of probability, where we must, therefore, assume a spontaneous development of the disease.

As to *a*. Here inherited tendency occupies the first place. The significance of this at the present day no longer needs to be proved by statistics and special instances, it is a fact noticed and conceded by all observers. The assertion may suffice that Echeverria out of three hundred and six patients under his own observation had eighty with a hereditary tendency. And in regard to this the idea of inherited tendency should not be taken in the limited sense, as if strictly epilepsy only in the ancestors led to the re-development of the disease in the descendants. Far otherwise, daily experience teaches us that the children of those parents also may become epileptic who have been mentally diseased, but who have never suffered from convulsive affections. The fact is, hereditary disposition must be taken in a far broader sense, and the proposition may be enunciated that any neurosis in the parents, whether it be of a lighter or more serious kind, may plant in the children the germ which may develop into epilepsy. This obtains not only in regard to hysteria, hypochondria, and catalepsy, but I have observed cases, where, *e. g.*, the mother suffered for many years from pronounced migraine, and with this exception there was absolutely nothing else in the way of family tendency to be found, and yet a daughter was hysterical, and a son epileptic; from my experience I am even inclined to ascribe to neuralgias of many years' standing in the parents a capacity of producing epilepsy in the children. There are even not wanting instances where, in connection with a mere "nervousness" of the ancestors, epilepsy made its appearance in the descendants. We must, however, also give special prominence to alcoholism, of the great importance of which in the causation of epilepsy, as well as of other nervous lesions, in the children of those who are victims of it, we can ourselves add a number to the already well-known and numerous examples. It seems to make little essential difference whether in all these cases diseases exist in the father or the mother ; it is not even necessary for it to be the parents directly ; a neuropathic tendency in the family generally suffices. Occasionally a generation may be skipped, and it may even happen that the progeny of epileptics remain perfectly healthy ; this latter is, to be sure, but rare, and the advice given even by Boerhaave, that epileptics had best remain unmarried, is decidedly judicious, if it could only be carried out in practice. It is proved by numerous examples that epilepsy also belongs among the various diseases from which the children of consanguineous marriages suffer.

That phthisis in the parents entails a tendency to epilepsy in the children, is in our opinion not correct, and if this relation often presents itself, it is certainly explained, as already observed by Hasse, by the actually very great frequency of phthisis. All the arguments brought forward again recently by Echeverria in favor of the connection between the two diseases lose their importance in view of this fact.

If epilepsy is hereditary, the first occurrence of its symptoms is generally early ; according to Echeverria, before puberty ; according to Reynolds, not later than the twentieth year. Our own experience agrees with the latter, and it may, therefore, in general be assumed that an individual with a hereditary predisposition, remaining healthy up to the twentieth year, will also escape epilepsy after that, so far as its development is excited by the inherited tendency alone. I will also add that such individuals often suffer from eclampsia as early as during the first dentition, but afterwards again remain apparently healthy up to the outbreak of the epilepsy.

As compared with inherited tendency, all the other influences which affect the organism or the nervous system as a whole are inferior in their capacity for exciting the central epileptic change. Such a capacity is ascribed to many influences, though, as already mentioned, of some it can only be proved with certainty that they caused the first seizure ; on the other hand, some seem to have been actually capable of evoking the epileptic change, although less frequently than is usually assumed.

Among the latter the habitual drinking of considerable quantities of alcoholics—*drunkenness*, in fact—assumes the first place; epilepsia potatorum has long been recognized. Apart from the complication with the usual symptoms of alcoholism in the intervals, this does not differ in its seizures from the ordinary form. The seizures are not always preceded by other severe symptoms of alcoholism, but sometimes even form the beginning of the series. At times the first seizure appears in conjunction with a severe fit of intoxication, but again at other times just when, for some reason, there has been a temporary total abstinence; in still other cases some external accidental cause occasioned the first attack.

It is worthy of mention that the various experimenters have been unable to produce epileptic seizures in animals by the continued administration of alcohol. We call special attention to the labors of Magnan,[1] who has studied this particular phase of the action of alcohol.

The older observers laid great stress on *sexual excesses*, and even announced coitum parvam esse epilepsiam (Ettmueller, Senac). Of late we have become much more sceptical in regard to this, and justly so.

The various reported cases where a first seizure has taken place during or as an immediate consequence of coitus certainly do not prove that the latter produced the epilepsy. Still, however, continued and great excess in Venere should be regarded as a veritable, though extremely rare, origin of the epileptic change. This is proved indisputably by a comparison of the abuse in Venere, which is so frequent, with the infrequency of epilepsy thereafter. Even the significance of masturbation was formerly decidedly exaggerated; and Herpin correctly observed that the causative relation is hard to establish when a vice is so widespread. At all events, its effects upon the nervous system must undoubtedly be more marked than even the excessive gratification of the sexual passion in the natural way. Nevertheless, examples of epilepsy after onanism are very infrequently recorded among careful observers. Among our own cases we have but one in which we could ascribe the origin of the disease to the vice we have mentioned. Echeverria insists that here *post* and *propter* may not infrequently have been confounded, as it is known that many epileptics first begin to practise onanism after they have had seizures. Still more should we doubt that absolute sexual continence may lead to the development of the disease. Although Tissot declared this to be an established truth, and even Herpin, in the case of women at least, would assign weight to continence as a "disposing" influence, still, all more recent observers, with the exception of a few, such as, *e. g.*, Radcliffe, from their experience, speak very cautiously in regard to this. In this connection we give a statement of Althaus[1] that congenital phymosis may lead to epilepsy through the medium of the masturbation which often accompanies it.

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[1] Besides various other places, compare Archives de Physiologie, 1873.

Close and continuous *mental exertion*, as well as powerful long-continued depressing emotions, especially grief, were in like manner formerly regarded as influences which could beget the epileptic change. On an impartial survey of the subject the conclusion must be reached that, in view of the frequent occurrence of the influences named, this is at least not proved; meantime we cannot wholly reject the possibility of it.

It is a question requiring further careful investigation whether certain general disturbances of nutrition, especially *scrofula* and *rickets*, to which epilepsy is often attributed, may occasion it, and particularly whether *an impoverished, insufficient nourishment* may have the same effect. The importance of the latter, in the development of an "active hereditary nervousness," has recently been again forcibly insisted on by Anstie,[2] and in addition to several older investigators, Echeverria is also of the opinion that cachexia in the parents gives rise to epilepsy in the children. And so calm an observer as Hasse thus expresses himself in regard to it, viz., that the disturbances of nutrition alluded to, "not infrequently seem to bring about a predisposition, in the presence of which proper exciting causes may lead to the outbreak of epilepsy." Very careful and comprehensive statistics only can determine this question.

Finally, there are instances well vouched for where real epilepsy has gradully developed itself from the *simulated* disease when of considerable duration.

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[1] The Lancet, 1867.

[2] The Journal of Mental Science, 1873.

If we, then, make a synopsis of what has just been said, the result is as follows: it is indisputably established with regard to a certain hereditary predisposition only, that it may develop in the subjects of it those changes in the central nervous system which constitute the essential characters of epilepsy. This is still open to question in a greater or less degree in all other assumed causes, and at all events their capacity for producing epilepsy is slight compared with the actual frequency of its occurrence.

As to *b*. With reference to another series of causes, we find ourselves upon a field much more capable of demonstration, for here we can in part plant ourselves upon the firm ground of physiological experiment. We are thinking of those cases in which definite anatomical lesions, acting upon some certain portion of the nervous system, induce epilepsy; that is, the development of the central epileptic change. The lesions may involve the peripheral nervous system, as is most often the case—the brain, or the spinal cord.

We have already expressed our views on this subject to the effect that we believe that the "reflex-epilepsy," occurring after *wounds of the peripheral nerves*, must be regarded as really the disease, and are of the opinion that the central epileptic change is induced in consequence of a definite excitation acting continuously from the sites of those lesions, exactly as in Brown-Séquard's experiments. What view should perhaps be accepted as to the connection will have to be touched upon further on. The lesion is then at all events an "occasional" cause; but it is in a special case the actual and only cause, which, without the aid of other influences, produces the disease.

Cases of the kind have already accumulated in such large numbers in monographs on the subject and in periodical literature that we may perhaps condense from them some general propositions. The nerves affected are either mixed or sensitive; most frequently it is the sciatic or the fifth, more rarely others, yet it may, as occasion offers, be any sensitive or mixed nerve. The lesions generally consist in external traumatic agencies acting upon the trunk, branches, or cutaneous distribution of the nerves, less often in pressure due to tumors or in neuromata.

In case of wounds of the skin, a peculiar cicatricial formation may sometimes be found.[1] The first seizure almost invariably appears some weeks or months, or even in some instances, years after the injury. The form of epilepsy which thus arises is generally marked by an aura of a sensitive, motor or vaso-motor character, proceeding from the region of the affected nerve. Sometimes, during the period which elapses between the receipt of the injury and the first fully formed paroxysm, there show themselves twitchings which are either limited to the territory of the muscles supplied by the nerve or are diffused over the whole corresponding half of the body.

In this category belong also those cases in which in women the disease receives its stimulus from the direction of the sexual apparatus. The importance of genital affections in this respect is excessively exaggerated; especially should those cases be excluded in which the menstrual period provokes simply the individual paroxysms, but not the epilepsy itself. Still, the latter also seems actually to occur. The connection in these cases must be regarded as wholly analogous to the lesions of other sensitive nerves, *i. e.*, a persistent anatomical lesion in the various parts of the genital apparatus acts through the medium of the sensitive nerve involved as a constant centripetal irritant.

I am inclined, however, to go even a step further in this direction. There are cases, of which several very pronounced ones have occurred in my practice, where absolutely none of the etiological influences mentioned, nor even any of the determining causes yet to be mentioned, can be found, and yet where a blooming child of from five to fifteen years is suddenly overwhelmed by a seizure, which is then repeated in the ordinary way. Now, in some of these patients it can be proved with certainty that they had suffered from eclamptic convulsions during the first dentition. I cannot get rid of the idea that here the process, at first purely functional, which was set up in the central parts at the time of the teething convulsions, may have furnished the impetus for the development of the epileptic change. Further on, in the chapter on Pathology, we shall touch upon the possible mode of development which may have taken place here. Still the foregoing view may be rejected in regard to some cases, and another held, according to which the connection existing between the dental convulsions and the subsequent epilepsy would be different from that which makes the former alike the evidence and the result of a nervous system already diseased. This question cannot be positively settled.

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[1] Compare *Schnee*, Two cases of reflex epilepsy. Zürich, 1861.

So far we have spoken only of lesions of sensitive, or at any rate of mixed nerves leading to epilepsy. It seems possible, however, for lesions of motor nerves also to be sometimes capable of this. At least several observations recorded in the literature of the subject point in that direction. But as this relation has not yet been determined with sufficient frequency and certainty, we content ourselves with this brief reference.

The above-mentioned experiments of Westphal and Hitzig (ll. cc.) have furnished experimental proof of the following proposition, which was formerly accepted clinically, but was subsequently doubted, viz., that *injuries of the skull, and correspondingly, affections of the substance of the brain*, may themselves lead to the development of epilepsy.

Hitzig's other experiments, in which irritation of the surface of the brain by strong electrical currents was followed by the outbreak of general convulsions, must of course be entirely disregarded in the inquiry as to the causation of epilepsy, although these experiments were repeated by Ferrier and others, and even made use of in theories of epilepsy. The reason is that in them there was only the outbreak of an epileptiform seizure during an irritation, but there was no later development of the epileptic state.

The *external injuries* which involve the skull need not by any means leave behind a lesion which is perceptible externally; on the contrary, such would not only be non-essential for the class of cases now occupying our attention, but would even bring the epilepsy into the preceding etiological group, providing it could be traced to this origin. Our interest is now much rather with a *cerebral* lesion induced by the violence directly, as in case of a hemorrhage, or indirectly, as when the cortical substance is wounded by splinters of bone. I can add another to the observations bearing on this point which are collected in the literature of the subject, and to which Leyden[1] has very recently added a case. Mine appears to me to be interesting in several ways, and especially as illustrating strikingly the experiments of Westphal. I only emphasize briefly the principal points.

A boy of eight years, who came of a perfectly healthy family, had never been seriously ill, and had had no convulsions in teething, fell a distance of twelve feet upon hard ground, striking on the head. He lay unconscious for some fifteen minutes, after which he roused, and at the end of ten minutes more was seized with a marked epileptic attack. There was a small scalp-wound on the right side of the head, which healed in a few days. After that for six weeks he was in a condition of perfect health; then there was again an epileptic seizure, and from that time they have constantly recurred, in former years at considerable intervals, of late years every four to twelve days. They always occur without an aura, being but very seldom preceded by a momentary dizziness. *They invariably begin by a turning of the head to the left;* consciousness is then first lost, the countenance becoming pale at the same time, and general convulsions declare themselves. Interparoxysmal symptoms exist to a limited degree; from time to time only a little headache; and sometimes there appear slight twitchings in the left half of the face or in the left arm. The patient, who is now a vigorous person of twenty-one years, appears to be of rather limited mental powers, and complains of a weak memory. Objectively there is nothing to be discovered except a cicatrix, about the size of a lentil, corresponding to the right coronal suture, and four centimetres distant from the median line. This is not painful or adherent, and when touched, either gently or quite roughly, no symptoms are manifested. No epileptogenous zone is to be found.

The fact that epileptiform seizures, recurring once or several times, may be *a symptom of different diseases of the brain*, such as diffuse anæmia or hyperæmia, incipient hemorrhages and embolisms, etc., has been already explained. There are undoubtedly cases, however, in which *a circumscribed focus of disease* becomes the starting-point of the epilepsy, exciting *the development of the epileptic change* in the medulla oblongata. In these cases the anatomical character of the original affection seems to be of less importance than its seat. Affections of the cortical portion seem to us of special significance in this respect. Thus it is a long-established rule that tumors, especially of the convexity, are accompanied, as they say, by epilepsy. It certainly is also true, in regard to many of these tumors, that they occasion *symptomatic* convulsions by their size or their mechanical relations leading to secondary anæmia, etc. Yet there are not a few cases where a very small tumor, the size of a hazel-nut, found at the autopsy in the cortex, has given origin to an epilepsy that existed for years.[1] The same thing is recognized in regard to exostoses, which affect the cortex, and also in regard to the remnants of hemorrhagic foci and centres of softening when similarly situated. As to the epileptiform seizures of paralytic patients with mental disease, Westphal[2] expressed the opinion several years ago that possibly they were not due directly to lesions of the cortex or ventricles, but to a secondary process in the pons or medulla oblongata.

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[1] Virchow's Archiv. Vol. LV.

In all such cases it must be assumed that the original lesion, in analogy with the injuries of peripheral nerves, has led secondarily to the development of an epileptic change. A more intimate knowledge of the method of this process completely eludes our view, just as the actual relations in their details still require the most careful study. We only consider the general way of looking at it just developed as in the main correct. We shall speak further on of the clinical peculiarities of this form of epilepsy.

According to Brown-Séquard's experiments, we might expect a rather frequent concurrence of *epilepsy with diseases of the spinal cord.* In reality, however, it is not so, provided we exclude what is in our opinion falsely called epilepsia spinalis (compare above, p. 198), and count only those cases in which the genuine central epileptic change has developed secondarily, in consequence of a disease of the spinal cord. Only a short time ago Westphal entertained doubts of the occurrence of instances of this kind, as did also Leyden;[1] yet recently several cases have been made known which scarcely admit of a doubt of their interpretation, as those, for example, which have been reported by Oppler,[2] Echeverria,[3] and Szontagh.[4]

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[1] Compare especially *Griesinger*, Cysticerken und ihre Diagnose. Ges. Abhandlungen. Vol. I. p. 399.

[2] Archiv für Psychiatrie und Nervenkrankheiten. Vol. I.

With regard to *c*. After excluding all those cases where the influences adduced under *a* and *b* have operated, in regard to which it can be assumed, with a greater or less degree of probability or certainty, that they were capable of bringing about the epileptic change, there still remain a considerable number for which not the slightest cause can be discovered; for, as to the influences presently to be discussed, which are often adduced as "causes" of epilepsy, under the operation of which the symptoms break out suddenly, we can allow such a designation to be valid only in exceptional cases, and even these may be entirely absent. It even happens that an individual, with no hereditary predisposition, etc., who has hitherto been in the full enjoyment of health, is very suddenly overwhelmed by a paroxysm in the night or in his sleep. In such a state of affairs there remains at present, in our opinion, but one assumption, viz., that *epilepsy may be developed altogether spontaneously, without any inducing influences whatever at present known.*

We remarked just now that many influences are mentioned as causes of the disease, of which it can only *with certainty* be said that they led to *the outbreak of the symptoms*, *i. e.*, that on their operation followed the outbreak of the first epileptic paroxysm. As to many cases, it seems to us unquestionable that a fright, or some other circumstance, merely called forth the explosion—sit venia verbo—while the peculiar epileptic change had long before been developed in the nervous system; in an analogous way, as an insufficiency of the valves of the heart may long exist, and palpitations, as a symptom of it, may not be observed until favored by some accidental occurrence, such as rapid walking or the like. Would any one, though, be willing to-day to say with certainty that this is always so? And if any one argues in this way: a severe fright provokes an epileptic seizure in a perfectly healthy individual, and the development of the epileptic change is excited only as a result of the unaccustomed functional processes which then take place in the medulla oblongata, we are not in a position to gainsay such a conclusion. We are here still moving wholly in the realm of hypothesis; but, on this very account, we regard it as advisable at present to characterize all these so-called occasional causes of "epilepsy" as such only when they induce the outbreak of the symptoms. This expresses the actual state of the case as at present known, and does not commit us to any false assumption. An extended investigation alone will be capable of bringing to a decision the question whether epilepsy can really come into existence in the manner indicated; and, even if this point were established, the *first* seizure would have to be regarded as only a purely functional process, based as yet upon no change in the medulla oblongata or pons, but which will itself furnish the stimulus for the development of such.

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[1] Klinik der Rückenmarkskrankheiten. 1874. Vol. I., p. 110.

[2] Archiv f. Psychiatrie und Nervenkrankheiten. Vol. IV.

[3] Loc. cit. p. 241.

[4] Wiener med. Presse. 1872. No. 5.

The mere number and the character of these assumed "causes" of epilepsy vary incredibly. Joseph Frank[1] brings forward over a hundred of them, but prudently adds, "which have excited epilepsy, *or*, *at least*, single seizures of it." It happens to every physician to hear it attributed to the most heterogeneous influences. Reynolds has arranged these in four classes, and distinguishes psychical or physical influences, eccentric irritation, and general organic processes. We believe we can, without disadvantage, omit a detailed enumeration; only the most important causes and those of most frequent occurrence will be selected.

At the head of the list stand *psychical* impressions of the most opposite character. Among them, fright again plays the most important part. Who could not bring evidence of this from his own experience? But also grief, anger, painful excitements, and joy belong in this category; in Tissot and Portal numbers of striking examples are pointed out. Terror is certainly the efficient cause in those cases where the sight of a person suffering from a seizure provokes the same in others; still, in these cases a pathological imitative instinct has also to be taken into consideration. One of the most striking examples of this is the well-known epidemic of epilepsy in the Haarlem Poor-house, reported by Boerhaave. Not merely an impression on the feelings, however, but also mental effort may provoke the outbreak of the first paroxysm, and even a repetition of it in the same individual. Our literature even tells us of cases where vivid dreams, especially if of a terrifying nature, have acted in the same way. Under my own observation, a girl had a first seizure from being obliged to eat the flesh of a cow that she had taken care of and been attached to.

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[1] *Joseph Frank*, Die Nervenkrankheiten. 1843. Vol. IV. Section on Epilepsy.

The attacks are not infrequently excited from the direction of the *genitals*. Some women are subject to them quite regularly at the period of menstruation, in which case they precede or follow the latter by a brief interval; and I have observed this especially in young girls, when the disease declared itself at the same time with the development of puberty. This feature, however, usually disappears after a time. It is known that in some individuals, male as well as female, the first coitus provokes a seizure, which is subsequently repeated every time. Anomalies of menstruation may also have the same effect. What is true of the genital apparatus is true also of *the organs of digestion*. In especial, overloading of the stomach with food, but, even more, the abuse of spirits on some single occasion, has been followed by a first seizure. In the case of a young merchant who was under my observation, in whom no other etiological incentive whatever could be found, the first paroxysms likewise followed upon an abundant meal, but thereafter without this exciting cause. Occasional diarrhœas have also induced the outbreak. Moreover, various observers have determined the beginning of the trouble during the course of or convalescence from *acute febrile* diseases; two cases of the kind have occurred in my practice—on one occasion after a pleurisy, on the other after measles. Intense *suffering*, in consequence of external injuries, or with internal affections, has sometimes brought about the appearance of the first seizure. Tissot and Radcliffe have maintained the same with regard to *over-exertion and great fatigue.* The latter author lays special stress on this influence, assuming, as does Tissot also, that it may develop the disease itself. With respect to this latter, Reynolds is able to adduce but one case in his own experience. In like manner, but one case has happened to me, which may perhaps be so explained. A soldier, who had previously been as sound as a nut, and who came of a very healthy family, was obliged during the Austrian campaign of 1866 to bivouac for eight days and to march a great deal. He then noticed a sensation of rigidity, a feeling of being asleep and numbness in the right arm, without lesions of motility ; this passed away after some days. Some weeks later, however, after further hardships in the campaign, he was seized in the bivouac with an epileptic attack, and this was subsequently repeated at greater or less intervals.

The influences just mentioned are those which relatively most frequently lead to the outbreak of the paroxysms ; with regard to many others, which are just as active on occasion and in individual cases, we refer to the instances cited in the works of Tissot, Portal, and especially Joseph Frank.

There yet remain to be mentioned some conditions which, without being capable in themselves of leading to epilepsy, yet occasion *a certain disposition to the development* of it, to such a degree that when it is present the special causes become more active. The importance of *age* in this connection is insisted on by all observers. In fact, this is so prominent that on some unessential points only is there any difference, while there is unanimity as to the main facts. We only adduce here the latter. Youth is the period of life which comprehends the beginning and development of the great majority of cases of epilepsy, and this is particularly true of the time between the seventh and seventeenth years, and indeed this proposition holds not only for cases where a hereditary tendency exists, but generally in all cases. It cannot as yet be explained exactly upon what this marked predisposition of youth depends. Of course we are inclined to point to the greater "convulsibility" of this period, and to think particularly of dentition, the development of puberty, and the development and increase of the whole organism. But it needs no discussion to show that this does not furnish the slightest explanation of this predisposition; we must simply be contented with the fact.[1] If now, on the one hand, the outbreak of epilepsy most often falls within the periods named, there is, on the other hand, no time of life at which it may not begin—from the first year to the seventieth (Reynolds) or the seventy-fifth (Heberden). It is not true that the period of involution in women acts in any way to favor it.

All other influences are subordinate to age; in fact it does not seem decidedly as if a special importance attached to any one. This is undoubtedly so with regard to temperament, the degree of intelligence, the occupation, and outward conditions of life. Still, in accordance with the accepted view, a definite influence ought to belong to *sex*, since women are more often subject to the disease than men. Several observers of the present day, however, arrive at exactly the opposite result, and Reynolds was able to determine no pronounced preference for one or the other sex. Our own view is in accord with that of Reynolds; we also found epilepsy about equally frequent in males and females. *Climatic conditions*, too, so far as is known, appear to be without essential influence on the development and frequency of epilepsy; it seems to occur everywhere. Joseph Frank, indeed, speaks emphatically of its frequency in Northern Russia and Poland. Meanwhile, as to the different regions of Germany (Königsberg in Prussia, Berlin, Breslau, Freiburg in Bohemia, and also Jena, so far as I can judge from a short period of observation), I can prove its quite uniform occurrence. Heat or cold seems to exercise no influence even upon the frequency of the individual seizures.

Pathological Anatomy.

Quite the same holds true to-day as when Schroeder van der Kolk said it nearly twenty years ago: "Gelijk in het algemeen de pathologische anatomie van zenuwziekten nog op een zeer lagen trap staat — — zoo is de pathologische anatomie en zijn de resultaten op het cadaver bij epilepsie nog wel de treurigste von allen." Not as if there were any lack of communications on alleged "characteristic" discoveries in the bodies of epileptics. Only a few of them, however, are of actual importance. Of course we shall not here detail all the post-mortem conditions which have been proved to exist in the cases of epileptics, and have been announced as the anatomical basis of the disease. With regard to some of them it appears to-day simply inconceivable how any significance at all could have been attributed to them, as, *e. g.*, to changes in the pituitary gland.

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[1] We have made an attempt at an explanation in the chapter on "Eclampsia," to which we refer the reader.

If we group together the post-mortem appearances, both macroscopic and microscopic, we find first, such as can be comprehended as standing more or less clearly related as a source of the trouble; second, such as appear rather as its results; third, such as with greater or less probability exhibit the proper anatomical basis of the disease; and finally, fourth, accidental complicating changes. As to several, to be sure, it still remains wholly uncertain to which group they belong.

There are very often found *irregularities in the structure of the skull* of various kinds, especially if the disease existed from youth or was hereditary. The most common of these is an asymmetry, more or less strongly marked, and which generally shows itself in a slightly marked paresis of the left side.[1] The bones of the skull are not infrequently much thickened and sclerotic, but by no means always so; and they may even remain normal though the disease last for a very long time, as I was able to assure myself this very day at the autopsy of a patient who had been affected with epilepsy for from forty to fifty years; in some cases, in fact, they are unusually thin. If now we take in connection with this the circumstance that *osteosclerosis*, with disappearance of the diploë, also occurs in cases of mental disease, existing for years without convulsions, there ought to be no doubt about the long received opinion that this represents a resulting condition, perhaps connected with repeated hyperæmias. Other irregularities occur occasionally, but by no means constantly, such as roughnesses of the internal surface, exostoses, narrowings of the carotid foramen, etc. *A stenosis of the foramen magnum* and of the beginning of the vertebral canal is of greater interest, and this originates either in diseased conditions of the atlas, axis, or occiput. A highly noteworthy case of this kind will be found quoted by Kussmaul and Tenner (loc. cit.); Solbrig[1] communicates nine of the kind and Hoffmann (loc. cit.) three others. There could be no question that the narrowing at this place by pressure, or by some other influence acting upon the medulla oblongata, produces the epilepsy, and is thus to be regarded as its indirect cause; it is equally certain, however, that in the vast majority this is not present, and consequently furnishes only an occasional cause of the disease.

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[1] *C. K. Hoffmann*, Vierteljahrschrift für Psychiatrie. 1869.

The *meninges of the brain* are sometimes normal, sometimes altered, in particular rendered opaque, thickened or distorted, the latter especially if osteosclerosis exists at the same time.

Various examinations have been instituted into *the weight of the brain*. Among the more recent authors Echeverria considers an increase of its weight to be an established fact, and looks for the cause of it in cerebral exudations and a proliferation of the neuroglia. It is not stated whether the psychical faculties had suffered during life in the eighteen brains that he weighed. Meynert,[2] on the contrary, found a decrease of weight in epileptic insanity, and, in fact, according to him, taking the whole brain, the greatest loss falls upon the cerebellum, while among the individual parts it falls upon the temporo-occipital portion. In the present state of our knowledge we can regard these changes in weight only as secondary, *i. e.*, unessential elements in the post-mortem conditions of epilepsy. Asymmetry of the cerebral hemispheres is more often found, but also occurs in the same way in other mental disorders.

Among the macroscopic changes of the brain—apart from accidental alterations—we first cite one which has made a considerable sensation. Meynert[3] ascertained the existence of a "disparity between the sections of the two hippocampi majores in epileptics, caused by the progressive atrophy of one of them, and this is coincident with callous or even cartilaginous hardness and marked anæmia of the same, not seldom with a waxy reflex and transparent appearance on section." Meynert himself, moreover, did not go so far as to locate the "seat of epilepsy" in the hippocampus major, as we have happened to read of in some places; he rather regards the affection of this part as secondary. This is not found to be constant, however, so that it cannot be essential to the disease; and even if it were so, we could make no use of the discovery at the present time, as the functions of the hippocampus major are still wholly unknown. (Compare my investigations on this subject, Virchow's Archiv. Vol. LVIII.)

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[1] Allgemeine Zeitschrift für Psychiatrie. 1867. Vol. XXIV.

[2] Vierteljahrschrift f. Psychiatrie. 1867. p. 125 et seq.

[3] Ibid., p. 396.

With regard to changes elsewhere in the brain, we should give prominence to those of which we have already said, in treating of the etiology, that they might furnish a cause for the development of epilepsy, such as small tumors of the cortex, etc. Finally, the rare cases are yet to be mentioned, where an abnormal distribution of the gray substance of the brain occurs in the cerebellum[1] or the cerebrum.[2] The significance of this anomaly and some of its relations to epilepsy are the less certainly established, and consequently explained, inasmuch as it also occurs without epilepsy.[3]

Since none of the macroscopic changes mentioned as found in the brain and its envelopes are constant, and as they are consequently non-essential, the more interest enters into the question whether *the microscopic investigation reveals regularly occurring changes in any one portion of the brain.* The answer, it seems, from the investigations especially of Schroeder van der Kolk and Echeverria, should be in the affirmative, and the latter authority comes to the conclusion that *in the medulla oblongata* constant changes can be proved microscopically to exist, while in other parts of the brain such are sometimes absent, sometimes present at the same time.

Schroeder van der Kolk reached the conclusion that at the beginning of epilepsy no organic change was appreciable. Subsequently an "albuminous intercellular" exudation shows itself between the nerve-fibres, which may lead first to induration and then to fatty degeneration and softening. Further than this, a dilatation of the capillaries, with thickening of their walls, is specially noticeable, in consequence of which the posterior half of the medulla oblongata appears more red and hyperæmic, whether patients have died in the seizure or not. The capillary dilatations lie chiefly in the region of the roots of the hypoglossus and vagus; more in the former, if the patients bit the tongue in the seizure, more in the latter, in case they did not.

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[1] *Meschede*, Bericht der Naturforscherversammlung zu Dresden. 1868.

[2] *Merkel*, Virchow's Archiv. Vol. XXXVIII.

[3] *E. K. Hoffmann*, Henle und Pfeufer's Zeitschrift. Vol. XXXIV. 3.

Different kinds of epileptics.	Hypoglossus.	Corp. olivare.	Raphe.	Vagus.
	Mm.	Mm.	Mm.	Mm.
A. Tongue biters.....	0.306	0.315	0.355	0.237
B. Not tongue biters..	0.210	0.217	0.300	0.348
Difference...........	+ 0.096 A.	+ 0.098 A.	+ 0.055 A.	+ 0.111 B.

Still, Schroeder himself expresses the opinion that these changes, which are of course established in the medulla oblongata in the later stage of the disease, presuppose a severe or even incurable character in it, but that they are dependent in their development upon a hyperæmia induced by the seizures themselves, and consequently are results of them, *i. e.*, do not constitute the essence of the disease.

Echeverria ascertained the existence of the same dilated capillaries in the medulla oblongata; besides these a granular albuminous exudation and granule cells, together with numerous corpuscula amylacea (not only in the neuroglia, indeed, but also on cross-section of the bulb); the ganglion-cells were also much pigmented in many places, especially in the nuclei of the hypoglossus and vagus. Analogous changes occurred in many cases likewise in various localities in the cerebrum, the ganglia at the base, and the cerebellum; but these parts were frequently wholly intact, the medulla oblongata alone showing changes in every case. It is also worthy of mention that Echeverria also frequently found the cervical sympathetic diseased more regularly even than the brain itself; the cells of the cervical ganglia, in fifteen cases that he investigated, were the seat of granular degeneration and filled with pigment, irregularly shaped, and their connective-tissue hyperplastic. Echeverria would attribute to these conditions the significance of primary, not secondary, changes, because he encountered them so constantly. It certainly is very questionable whether any importance whatever can be ascribed to the conditions described by Echeverria, especially those of pigment in the nerve-cells, since A. Labimoff[1] has shown that the pigmentation of the sympathetic nerve-cells is found as a regular condition in persons advanced in life, and not so very rarely even in young persons, who have died of diseases quite distinct from epilepsy.

Without considering detached investigations, let us here mention the points discovered by L. Meyer.[2] This author ascertained, like Schroeder and Echeverria, a diseased condition of the vessels, particularly a wide-spread fatty degeneration, sclerosis with localized obliteration, and in isolated places also small dilatations on the smallest arteries and capillaries. These conditions were met with in the medulla oblongata, the cortical substance of the cerebrum, and the uppermost part of the cervical portion of the cord. Meyer, however, is likewise inclined to regard the affections of the vessels in many cases as secondary. And the principal thing is that they are not at all characteristic of epilepsy, for they are also met with in progressive general paralysis and other diseases.

If we review the anatomical results, the showing is certainly a very poor one. No alteration is shown with certainty to be constant. Even with regard to the changes in the bulb it is questionable whether they are primary or secondary. There is only one thing to which the investigations now before us point, viz., that, generally speaking, the primary histological departures from the normal are to be looked for with the greatest probability in the bulb of the medulla.

Symptomatology.

The character of the clinical picture of epilepsy is chiefly impressed on it by the nature of the individual *seizures* designated as "*epileptic.*" Besides the paroxysms, patients not infrequently also present *symptoms belonging to the interval*, which sometimes may equal in intensity those belonging to the paroxysms or even exceed them. The symptoms of the intervals, however, are never distinctive of the trouble as epilepsy in any special case; that, we repeat, depends on the paroxysms only.

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[1] Beiträge zur Histol. u. pathol. Anat. d. sympath. Nervensystems. Virchow's Archiv. Vol. LXI.

[2] Archiv f. Psychiatrie u. Nervenkrankh. Vol. III.

I. *The Epileptic Seizures.*

For a long time various forms have been distinguished according to the character of the attacks.[1] At the present time certain expressions only will be retained in this connection, for the sake of readier comprehension, because they are associated with long-received ideas. We are inclined to adhere to them chiefly in the interest of a clear demonstration, because the great abundance of material bearing upon the symptoms actually demands an appropriate classification. Certain of these forms occur more, others less frequently, and at the same time there are the various transitions between them. The determination of them is naturally in part an arbitrary one. We make choice of the following:

1. Epilepsies in which the classical paroxysms make their appearance with coma and general convulsions (*E. gravior, haut mal*).

2. Epilepsies in which paroxysmal loss of consciousness alone occurs, the spastic element, for the voluntary muscles at all events, being absent (*E. mitior, petit mal*).

3. Epilepsy in which, with inconsiderable loss of consciousness, partial twitchings occur in the regions of certain muscles, whereby in the most various ways a transition is effected between the cases mentioned under 1 and 2.

4. To these we add the irregular forms of seizures and the epileptoid states.

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[1] Thus, *e. g.*, the following classification is found in *Peter Frank* (loc. cit.): 1st. Ratione accessionum: *a*, E. vaga; *b*, periodica. 2d. Ratione extensionis: *a*, E. universalis; *b*, partialis. 3d. Ratione modi concidendi: *a*, caduca; *b*, cursoria; *c*, gyratoria, etc. The triviality of such a classification is self-evident.

A. *Epilepsia Gravior.*

The seizures either set in with extreme suddenness, or they are preceded by warnings.[1] As it is very difficult to furnish complete numerical data about the occurrence of the latter, and as such numbers in our opinion would be destitute of a special scientific value, or, at all events, would throw no light upon the pathology of the trouble, we content ourselves with the approximate statement that prodroma show themselves in somewhere about half of the cases.

These *premonitory symptoms* of the paroxysms have long been divided into remote and immediate. For the latter the term "aura epileptica" is still extensively in use, and this may also be retained, provided we have a clear conception that an aura, in the old sense, *i. e.*, the sensation of a breath blowing upon the patient, scarcely ever occurs, and that consequently this expression in its transferred meaning should only convey the same signification in general terms as warnings.

The *remote warnings* all observers agree to be very much rarer than the immediate, and this I, too, must confirm. At the longest they occur from two to four days beforehand. In these cases they almost always show themselves under the form of psychical changes. Patients become sad and of an oppressed frame of mind; they retire into themselves, or, on the contrary, they become excited and talkative, violent and irritable, or quarrelsome and distrustful. At the same time they complain of dizziness, headache, and confusion of the head; sometimes these symptoms even form the only prodroma. After these have lasted for one or several days, the seizure breaks out, and afterwards the old condition belonging to the interval returns. These remote warnings show themselves in individual patients especially when they have remained free from paroxysms for a considerable time. But a real seizure does not always follow upon them; sometimes such a one is confidently expected as usual, and yet it remains absent. Much less often than with the mental symptoms mentioned do we meet with others. Reynolds mentions that he has sometimes seen a peculiar dark coloration of the skin, especially in the face and neck, as much as twelve hours before the seizure. A lady under my observation is able to predict the seizure with certainty every time. While usually her sleep is light and rather short, she then has a very deep and continued sleep. Still she awakes in the morning perfectly well, but in the course of the day the paroxysm breaks out. Certain patients also feel for several days beforehand a leaden weight in the limbs or a slight trembling.

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[1] In order not to interrupt the demonstration according to symptoms, we speak here of "warnings," following the established custom. In what light we regard their relation to the seizure proper, we will state in the section on "Pathology."

The *immediate prodroma*, the real aura epileptica, exhibit a considerably greater variety than the remote. The question whether they should even be looked upon as symptoms of the seizure itself may temporarily be left out of consideration. This aura may begin in the territory of the most different nerves, although more often in certain ones than in others, in the sensitive, motor, vaso-motor, and also the nerves of special sense. We cannot possibly enumerate the extraordinary variety of its manifestations in every individual case; we must content ourselves with a general outline.[1] The *sensitive aura* is characterized by tickling sensations, or by dragging, tearing pains which seldom extend in the definite course of a nerve, but generally over a whole extremity, and, in fact, usually from the periphery, the toes or tips of the fingers, upwards towards the head. In other cases patients are affected with general or one-sided headache; in others again with violent pains in the epigastrium with or without a tendency to vomiting, and this in particular Hasse has often observed in the female sex. The opposite also occurs, that is, a decrease of the sensory function, whereby the patient suddenly or more gradually experiences a loss of feeling in one extremity or one-half of the face. In one of my patients this sensation always began on the right side of the face and head, extended downwards from the shoulder over the right arm, as well as in a centrifugal direction over the corresponding leg, and finally over the right half of the body too. In this case as well as in some others where the phenomenon preceded the seizure a sufficient time, say ten minutes or longer, I could make out a decided diminution in the sensitiveness to the prick of a needle and to the impression of temperature. I have observed a *vaso-motor* aura scarcely less frequently than the sensitive,[1] and I am persuaded that many cases must be ranked with the latter which were formerly counted among those of sensitive aura. In this case patients complain of a feeling of numbness, generally beginning in the fingers or toes of one extremity, accompanied with decided tickling and a sensation of coldness and weight. While this is going on, the affected parts become very pale and cold to the touch, and blunted to slight external impressions. These symptoms also take a centripetal course. The phenomenon might also be referred to a participation of the nerves of the vessels if the attack, as sometimes occurs, is preceded by shivering or transient burning heat. Certain patients get a redness in spots in various parts of the body. The *motor* aura is variously displayed, ordinarily as a spasm in the region of certain muscles or groups of muscles, which is generally of a clonic, more rarely of a tonic, character, sometimes also as a trembling or shaking motion. Here, too, the extremities are affected most frequently, then the muscles of the face, the orbicularis palpebrarum, other muscles much less frequently, *e. g.*, those of the ear (Romberg), the sterno-cleido-mastoid, etc. It belongs among the decided exceptions when the seizure is ushered in by the opposite condition, the paresis or even complete paralysis of an extremity (I have myself seen two cases of this kind), and in regard to such cases it is always doubtful if we have to do with a genuine epilepsy. (See further on under the remarks on Epileptic Palsy.) Now and then spastic phenomena in the region of the internal organs show themselves as a prelude to the paroxysm; thus rumbling in the belly, straining at stool and on passing water, drawing up of the testicle, palpitations, etc.[2] Besides these, the literature of the subject recognizes a great many instances where symptoms referable to the *nerves of special sense* preceded the seizure. The optic and auditory nerves are relatively the most frequent participants in these cases, which are in any event quite rare; the olfactory nerve is much more seldom involved.[1] Patients have impressions of light and color, and even at times the hallucination of corporeal figures; they hear sounds and voices; they experience sensations of smell which are of a predominantly disagreeable kind. Joseph Frank saw a patient in whom the attack announced itself by sensations of a sweet taste. In very isolated cases changes of *secretion* also develop as prodromata, as *e. g.* a profuse secretion of tears or perspiration (the latter I have several times observed) or an abundant flow of saliva. Most often, undoubtedly, and this accords with my own experience, *cerebral* or mental phenomena form the immediate warnings. It is a fact that many patients are met with who state that they are "dizzy" immediately before the onset of the seizure. It can certainly be properly assumed that under this term something else is often understood, such as overclouding of the senses, confusion, etc. Besides this "becoming dizzy," other symptoms, such as sudden excitement, loquacity, and the like, form but seldom the immediate prodroma. Finally, an aura occurs now and then which can be brought into no one of the classes mentioned. There are very indefinite feelings of general restlessness, general discomfort, peculiar sensations in various parts of the body, about which patients can give no more accurate information.

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[1] Any one who is interested in knowing where to get a knowledge of remarkable and peculiar warnings will find a collection of such in *Joseph Frank*, and particularly in *Delasiauve*, loc. cit., pp. 21-26.

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[1] Farther on, under Pathology, I will enter more fully into the peculiar form described in recent times as Epilepsia Vasomotoria.

[2] The statical conditions, which have been observed as prodroma in very rare instances, will be considered in another place.

The aura does not always show itself exclusively in the sphere of one nerve or another; it is not so very unusual for the sensitive to be combined with the motor, or the latter with the vasomotor, or for dizziness to be associated with one or more of those just named.

The duration of the aura varies within very fluctuating limits; at one time it may last for two hours, and then again it runs its course with the rapidity of lightning; a sensation, of which the patient afterwards says that it ran up in an instant from the finger-tips to the top of the head, a scream, and the patient is thrown upon the ground. On an average, however, the aura, when present, generally lasts from half a minute to five minutes.

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[1] Compare *Sander* in the Archiv für Psychiatrie und Nervenkrankheiten. Vol. IV.

It happens not at all infrequently that the customary aura—as we pointed out in regard to the remote warnings too—shows itself, the patient expects a seizure, and yet it keeps off. This may even take place several times in the course of a day, especially in epileptics who suffer from rather frequent and pronounced paroxysms recurring every few days. Every one who has seen many epileptics will recall such as these, in whom there often appeared dizziness or some slight twitching in an arm or one side of the face, or tickling in the leg, and yet a veritable seizure only occasionally succeeded these manifestations. One of my patients had a vaso-motor aura in one leg from six to ten times daily, and yet only on an average every week an attack following upon it.

It will depend essentially upon the view which is held regarding the aura, what significance is ascribed to the circumstance last mentioned. We anticipate here by saying that, in our opinion, these abortive auræ, which are indeed free from obscuration of the senses or any sensation of dizziness, are to be placed in the same rank with what is called "*petit mal.*" We shall return to this question.

The literature of the subject has various undoubted instances where, when an aura has lasted quite a considerable time, the seizure has been successfully warded off. If a dragging sensation mounts gradually from the fingers to the top of the head, and we rapidly tie a ligature firmly about the upper arm, or if, in case of a spasmodic flexion of a finger, this is forcibly straightened, and so on, we may even keep back the attack. From these particular occurrences has evidently developed the absurd popular belief in the advantage of bending back the thumb in the seizure itself.

To settle this point, we remark that in all probability, as Brown-Séquard also assumes in his experiments (see above), it has nothing to do with the repression or breaking up of a process of excitation which is creeping towards the centre. The phenomenon is rather to be viewed as in the nature of a "reflex inhibition." We must here, of course, refrain from a discussion of the question of what we are to understand physiologically by this reflex inhibition, and only add that results, in their nature analogous, have been obtained experimentally by Setschenow,[1] Herzen,[2] Goltz,[3] and myself.[4]

Now, whether preceded by prodroma or not, the striking symptoms of the seizure itself, with its two essential leading characteristics of *loss of consciousness and general convulsions*, almost always break out suddenly in overwhelming force all at once. Before the patient falls to the ground, or perhaps at the same moment, he often utters a cry, as Romberg expresses it, "shrill and terrifying to man and beast." This cry is not, however, characteristic; at least it is absent more often than present. We regard it as unimportant to bring forward fuller figures as to the frequency of its occurrence.

Much has been written on the significance of the epileptic cry. So much as this should be considered as settled that it is not to be regarded as the effect of any mental action whatever, such as pain, fright, or surprise, as Herpin and others thought, albeit in very rare cases patients are not at the moment wholly unconscious. Still it is generally uttered simultaneously with the loss of consciousness; and we entirely agree with the conclusion that it is simply of spasmodic origin, *i. e.*, the product of a spasmodic action of the respiratory and laryngeal muscles, and either of an inspiratory or expiratory nature.

Innumerable accounts, more or less true to nature, have been written of the epileptic seizure; some are distinguished by an almost dramatically excited vividness in the description, as above all, those of Romberg and of Esquirol, also those of Tissot and many others. Obviously the disease itself almost provokes such a description with its shocking, violent symptoms, the animal roar, the sudden downfall of the affected person all unconscious, the terrifying spasmodic movements. Still, at the expense of fine language, we prefer a quiet statement in the interest of scientific clearness.

In individual cases, and, in fact, in particular seizures of the same patient, manifold diversities are found, even in the features of the full epileptic attack; these will be attended to hereafter.

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[1] Ueber die elektrische und chemische Reizung der sensiblen Rückenmarksnerven des Frosches. Graz, 1868.

[2] Expériences sur les centres modérateurs de l'action réflexe. Turin, 1864.

[3] Functionen der Nervencentren des Frosches. Berlin, 1869. p. 39 et seq.

[4] Zur Lehre vom klonischen Krampf. Virchow's Archiv. Vol. XLIX.

In the first place we want to sketch the type of a seizure as it is most often displayed.

Apart from the prodroma we must distinguish *two periods of the attack;* then there must be added as the third an *after-stage.* While the loss of consciousness is the same in both, they are principally to be discriminated by the different character of the spasms; in the first period it is tonic, in the second clonic.

First period.—Consciousness is completely lost. At times the patient falls down as if struck by lightning; instantly and as if by a stroke he is deprived of all mental functions. This comes so quickly that he falls headlong in any attitude or place quite regardless of his surroundings. If the convulsion does not declare itself at the very same moment, he falls like a dead mass, evidently in consequence of the sudden relaxation of all the muscles; still this condition of things happens extremely seldom. At other times the loss of consciousness comes on more gradually, as in the course of a few seconds, and the sufferer gains time enough to assume some recumbent position voluntarily. Generally, however, the arrest of all mental activity takes one so by surprise that from the beginning of the seizure no recollection is retained of the facts of the case. It is not necessary to draw attention to the fact that in deep coma every conscious sensation is wanting; it is well known that this goes so far that patients have sometimes fallen into the fire and burned themselves. But even reflex processes are absent in many cases; the iris does not contract under the stimulus of light, the lids are not closed when the conjunctiva is irritated; at other times, again, reflex processes are present; the lids close, and the limbs are drawn together when cold water is dashed upon the body (Romberg).

The tonic spasm now declares itself. Its extent is variable; in the severest cases it involves the whole muscular system. The pupil is fixed, sometimes appearing to have a pronounced expression of some mental emotion, anger, etc., the countenance distorted, the jaws closed upon each other, the head drawn to one side, or backwards; there is general opisthotonus with marked extension of the extremities (with an astonishing spreading asunder of the fingers and toes, or also very great incurvation of the foot—Tissot), sometimes emprosthotonus, or even—in fact, quite frequently—one half of the body shares in this to excess with curvature in that direction, and with an alternating tendency to one or the other side; tonic contraction of the muscles of the throat (trachelismus—M. Hall), of those of respiration (with absolute arrest of breathing), and of the laryngeal muscles. In a child four and a half years old, that was under my observation there followed upon the cry, uttered when consciousness was already gone, a sharp, strong, whistling inspiration lasting for some seconds, such as is observed only in pronounced spasm of the glottis, and then the clonic spasm.—The tonic influence, however, is not always so general and so strong; sometimes it is limited to certain groups of muscles only, sometimes but very slightly pronounced. And if it is general, certain regions may be attacked rather than others; most frequently it is first marked in the muscles moving the head and in those of the globe of the eye and the face.—As already stated, the tonic spasm sets in either simultaneously with the coma, or rarely somewhat later, or it may even begin a little earlier; in the latter case the scene opens with fixation of the eyeball, distortion of the face, and turning of the head. We likewise remark here that occasionally the tonic spasm is wholly wanting, and the scene begins at once with clonic twitchings; but the opposite also occurs in rare cases, namely, the convulsive element is indicated by a tonic fixedness only, in which the rigidly extended extremities at the utmost undergo only a slight shivering movement. The latter point will be more fully discussed hereafter.

The coloration of the face changes. While the earlier observers spoke almost exclusively of a dark cyanotic discoloration, later ones report pallor just as often. Both are correct; both changes occur, and, in fact, generally in the same patients one after the other. My observation agrees with that of Radcliffe, Sieveking, and Brown-Séquard, that in the majority of cases the patients grow pale at the beginning of the seizure. Sometimes its approach is indicated in this way: the countenance changes before the loss of consciousness or the tonic spasm is developed; at other times all begin at the same time. The pallor then lasts during the whole of the first period, or it is present at the very beginning only, and even before the clonic stage has declared itself the face has already acquired a dark tint.—Still it must be remarked that sometimes the hue of the face remains unchanged during the whole initial period, and at other times it is dark-red from the very beginning. The latter appears to be the case particularly if just at the beginning a decided spasm of the muscles of the throat prevails with compression of the jugular veins and spasm of the glottis.—In recent times attention has also been directed to the condition of the fundus oculi. It is obvious that an ophthalmoscopic examination during the convulsions is hardly practicable. Still we have some observations made during the prodromal stage (Echeverria and others) which show that sometimes, but not always, anæmia of the fundus is present. Unfortunately it is not stated how the coloration of the face behaved in these cases.—The iris is generally dilated at the beginning of the attack.

The state of the pulse is variable. In certain cases it remains unchanged both during the first period and throughout the whole attack. At other times no examination at all is possible during the convulsions. According to other observers, however, it is "small" during the tonic stage, *i. e.*, the radial artery is somewhat narrowed and the wave reduced. With regard to tension and regularity, the reports of various authors again differ very much. It may happen that the pulse at the wrist is not felt at all, while the carotids are beating and the action of the heart is normal.

Echeverria gives drawings of sphygmographic curves, according to which the pulse before the seizure was higher, distinctly dicrotic, and accelerated.

The tonic stage is generally of short duration, often only a few seconds long, so that it is overlooked by the laity in consideration of the performance which now follows it, or it may last from one-quarter to one minute. Upon this then follows the

II. *Period of clonic spasm.*—The profound unconsciousness continues wholly unchanged; but instead of the tonic muscular contraction the most violent convulsions now set in, the form of which is so well known that it is used as the type in describing analogous spasms arising from the most different sources. We may spare ourselves a detailed account, and call attention only to the following: almost all the voluntary muscles of the extremities, trunk, and head take part in the epileptiform twitchings. Through these are brought about in the full seizures positions and attitudes of apparently the most impossible kind, which vary with surprising rapidity. The violence is sometimes so considerable that the most dangerous wounds are received; fractures and dislocations have been observed, breaking of the teeth or portions of them, deep laceration of the tongue, and rupture of muscles, not to mention the wounds and excoriations of the skin which arise from the headlong way in which the body is thrown about. Very often the clonic jerkings are interrupted one or more times by tonic spasm, so that the patient again becomes rigid; or while certain parts are undergoing violent clonic spasmodic movements, others are in a state of fixed tonicity. Even if the clonic action is very general, it may still very often be evident that one half of the body takes a more active share in it than the other. Usually, but not always, saliva shows itself at the mouth of the sufferer in the shape of foam, which may be tinged with blood from wounds of the tongue and buccal mucous membrane.—Increased peristaltic action of the intestines occurs with rumbling in the belly and discharge of flatus and fæces, sometimes tympanites, probably from air that has been swallowed, also ejaculation of semen, rarely vomiting, and occasionally escape of urine with so much force even as to rise in a stream five or ten feet high (Tissot, Portal).—The respiration is forcible and quickened, while again it may be spasmodically interrupted. The consequence of this and of the violent action of the muscles is a marked venous hyperæmia. The jugulars are swollen up, the face acquires a deeply cyanotic color, and the eyeballs protrude. Sometimes subcutaneous vessels are ruptured with the formation of ecchymoses in the skin, especially in and around the eyelids, as well as sometimes in the internal organs, yet we have noticed that cerebral hemorrhage forms one of the very rarest accidents in connection with the epileptic seizure. The older observers speak of discharges of blood from the rectum and vagina, and of bloody tears.—The pulse, provided it can be tested during the violent action of the muscles, is now fuller than in the first period, and frequent.

After this wild scene has lasted a variable time, from half a minute to three minutes, and only in very exceptional cases longer, the time of course *appearing* to bystanders very much greater, the *after-stage* of the seizure is developed. The convulsions either stop suddenly or wear off more gradually, and the patient either lies with his limbs completely relaxed, if the sensorium still remains profoundly affected, or the muscles at once recover their normal tonicity. Here and there isolated supplementary jerkings still occur, or perhaps a tremor runs over the whole body.—The return of consciousness takes place in various ways. As a rule, the patient still lies in a deep coma for some minutes after the cessation of the spasms. Then he opens his eyes, wakes as if out of a deep sleep, looks confusedly around him, closes his eyes again, mutters something incomprehensibly, groans, sighs, or even speaks, but all as if his mind were wandering. At this period he also responds to any considerable sensory irritant, or when called to. The distressing cyanosis diminishes. The iris contracts. The respiration grows more quiet, deeper, ordinarily uniform, though sometimes in a measure intermittent, or at all events irregular in rhythm and depth ; after violent and prolonged convulsions râles are heard in the chest, which evidently arise from a considerable accumulation of fluid in the bronchi, caused by the venous stasis during the spasmodic stage. The pulse becomes gradually more quiet and full; Echeverria indeed demonstrated with the sphygmograph a decided decrease in the height of the pulse-curve during the comatose after-stage; it only became higher again after from ten to fifteen minutes. Voisin[1] states that the ascending branch of the wave of the pulse is higher than normal, and that the curve is characterized by a pronounced dicrotism—appearances which outlast the seizure by from half an hour to several hours. The skin is soft and often covered with an abundant perspiration ; sometimes the discharges spoken of above only now make their appearance. We will add here that ophthalmoscopic examinations after the seizure have shown a marked hyperæmia of the fundus oculi, which may last for twenty-four hours.

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[1] Annales d'hygiène publique. 1868. Avril.

Thus a quarter of an hour after the seizure the patient may find himself wholly in his previous condition, sometimes without the slightest bad results. There only remains the deficient recollection of all that happened to him in the moments just past. Only seldom, however, does the seizure pass off so rapidly and leave so few traces behind. As a rule, the after-stage lasts longer; perhaps a somnolent state follows upon it, or an abnormally deep sleep, the duration of which varies from half an hour to several hours; and if the attack was quite violent, the patient feels fatigued, unnerved, and as if bruised all over. The mental tone is altered at times, and becomes depressed and irritable. Still occasionally, especially when psychical prodromata of considerable duration have preceded it, patients feel relieved after the seizure, are freer mentally. It occurs exceptionally that the sleep after the attack lasts for many hours, even for twenty-four, before patients are again completely masters of themselves; and in two cases under my observation the somnolent condition extended over two days. Some other sequelæ, which occur much less frequently, must be mentioned later, after the other forms of the seizures are described.

The temperature of the body after the epileptic attacks has been measured by various authors, without a complete agreement having yet been arrived at; of course it is understood that we are here speaking of single seizures, not of such as rapidly follow one upon another, the *état épileptique* of the French. Williams,[1] while considering his efforts as only slightly successful, concludes that after strong convulsions, especially after tonic spasm, the temperature may rise as much as 3° F. Clouston expresses himself rather indefinitely, and moreover, Bourneville[2] recognizes his procedure as defective. This latter, and also Voisin, found a very slight elevation, say a few tenths of a degree. Westphal,[3] with whom we agree, made up his mind that an elevation of temperature after the isolated seizures of epileptics is exceptional.

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[1] Medical Times. 1867. Vol. II. p. 225.

[2] Études cliniques et thermométriques sur les maladies du système nerveux. 1873.

[3] Archiv für Psychiatrie u. Nervenkrankheiten. Vol. I.

The changes that may take place in the urine evacuated after the seizures as to its quantity and quality have attracted special attention since Reynoso and Heller maintained that sugar was temporarily present in it after the attacks. With rare unanimity has this opinion been contested; Michéa, Delasiauve, Sieveking, Hasse, Reynolds, Echeverria (with the exception of one case), Ebstein,[1] M. Huppert,[2] I, and various others, have never been able to discover sugar. Probably the earlier accounts resulted from errors in the methods of examination. The same negative conclusion has been reached by most of the authors named with reference to the occurrence of albumen. On the other hand, a considerable conformity of views prevails as to this, viz., that after the attack a quantitative change sets in so far as that the amount of urine is decidedly increased, and at the same time of a clearer color, while its specific gravity is not essentially altered. Ebstein, indeed, was able to arrive at no constant results as to the increase of urine. According to several observers, such as Parkes, Gibson, and Echeverria, the amount of urea is at the same time increased, generally also that of the phosphates, while no increase of the uric acid could be proved.

M. Huppert (loc. cit.) asserts recently that *every* fully formed epileptic seizure is immediately followed by a distinct transient discharge of albumen in the urine, lasting from two to eight, generally from three to four hours. For the precautions to be observed in the determination of the albumen we must refer to the original article. Vertigo usually remains free from appreciable albuminuria. Huppert, in addition, ascertained the startling fact that in about half the cases of pronounced attacks, though less often after undeveloped ones, the urine first passed contained, besides albumen, hyaline cylinders and quite numerous spermatic filaments. The cylinders disappeared sooner than the albumen, for in the second urine passed they were but very seldom encountered. After mere epileptic vertigo, cylinders and spermatic filaments were both absent.

The cases which I have examined since the appearance of Huppert's work on albuminuria, have not yet been numerous enough to enable me to give an opinion. Still I may even now say that I have occasionally found the reaction for albumen faintly declared.

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[1] Deutsches Archiv f. klinische Medicin. Vol. XI.

[2] Virchow's Archiv. Vol. LIX.

B. *Epilepsia Mitior.*

Formerly only the full paroxysms just described, and those to be mentioned hereafter, which were accompanied by more or less pronounced local spasms, were characterized as epileptic; in the presence of these only was the disease assumed to exist; but fuller observation has taught us the fact that epilepsy may manifest itself in the form of seizures, whose *only characteristic* is a *loss of consciousness*, without any visible outward spasmodic element, or at most accompanied by fixation of the eyeball. At the present day it requires no detailed demonstration to show that these and the forms described under C, are actual epilepsy. In former times they were often ascribed to "apoplectic congestion of the brain," an abuse against which Trousseau in particular warmly protested. Still I consider it as equally injudicious to designate these forms as "epileptic dizziness." There is almost without exception more than a dizziness; there is complete arrest of consciousness. We shall return later to the matter of epileptic dizziness proper.

The time during which unconsciousness lasts is very brief, generally only from a few seconds to half a minute, yet it may even continue for from four to eight minutes, as I have convinced myself. As a rule, consciousness is lost without any premonitions: while eating, the patient lets his spoon and knife drop, his look becomes fixed; sometimes this lasts only so long as to excite the attention of those around him; then he resumes eating again. While in the midst of talking, he suddenly stops; after a brief absence of mind he goes on again. While walking upon the street he stops still, but need not necessarily fall if the arrest of consciousness is brief enough. It may even happen that the more automatic actions are not once interrupted, the sufferer goes on walking, continues to play on the piano, and the like. But, on the other hand, when the absent-mindedness is longer, he may fall down, or, if riding, be thrown from his horse. At times such a patient is thrown down suddenly and lies there unconscious, until he shortly rouses himself; an inexperienced person would be disposed to look upon the attack as a paralytic stroke, although in apoplexy the overthrow of the brain scarcely ever happens so suddenly. (Compare Cerebral Hemorrhage.)

As already observed, these seizures generally overtake one without warning; yet the most various auræ are also sometimes noticed. The most common warning, in my observation at least, consists in a sensation of dizziness, in flashes, and darkness before the eyes. Reynolds asserts that the color of the face in these seizures may on occasion show all the variations mentioned above, of course excepting cyanosis; in my patients a sudden pallor could generally be made out, which both preceded and outlasted the loss of consciousness.

While in the majority, to be sure, after-effects are wholly absent, so that patients are quite unaware that anything whatever has just happened to them, at other times, even after slight attacks, symptoms show themselves which last for some time, it may be for several hours—in most instances a suppressed voice, bad humor, and surliness; or headache and forgetfulness, or depression and fatigue with a tendency to sleep. We shall have to discuss further on how these conditions are to be distinguished from simple faintness, with which they have not very infrequently been confounded by inexperienced observers.

C. *Transition-Forms; Loss of Consciousness with local Spasm.*

Epilepsia mitior in the form just described, *i. e.*, complete arrest of consciousness without any spasm visible externally, certainly belongs to the exceptions. On the other hand, we quite often meet with transition-forms between it and the major attacks, in which slight spasms occur together with coma. For the description of these attacks we may in the main simply refer to epilepsia mitior, especially as concerns the duration, the after-effects, and the loss of consciousness. To these, however, spasmodic phenomena now come to be added.

The locality, the intensity, and the nature of these are subject to the greatest variation. Trousseau gives a very lively description of these so-called abortive attacks, and mentions various examples in illustration.

It happens but seldom that tonic and clonic spasms appear together or in succession as in the major attacks; as a rule, there is in this form only one or the other kind. All the various possibilities of the picture so constituted, which come under observation, cannot be detailed; a brief indication of them must suffice. In one patient the spasm betrays itself only by firm closure of the eyelids, in another by strabismus, or by twitchings of certain of the facial muscles, or by violent contortions of the face, movements of the lips, shutting the jaws firmly, chewing motions, rolling the tongue back and forth, a constrained turning of the head in some direction, or shaking movements of it. Reynolds frequently observed arrest of the respiration by spasm of the external respiratory muscles and the diaphragm, and in consequence slight cyanosis. Together with these symptoms on the part of the muscles of the head and back, or even without these, there may occur local spasms in the extremities, or, less often, in the trunk; certain fingers are rigidly bent or stretched; one arm, one leg, or even two extremities become stiff; or the parts mentioned are moved to and fro by rapid clonic twitchings. Or a slight tremor runs over the whole body. Compare, too, the section further on about Epileptic Hemiplegia.—Again, there are cases which are closely allied in their external features to the major attacks, inasmuch as the whole body is subject to slight tonic and clonic convulsions, which, however, from their very slight degree of intensity and their very short duration, rather suggest to the novice an analogy with the pronounced attacks than prove it. It is quite unprofitable to undertake to enumerate here all the possible multifarious varieties of the picture; the reality surpasses any description.

In the case of epilepsia gravior the unconsciousness always outlasts the spasms. This may also be the case in the form which now occupies our attention, though the opposite frequently occurs here, *i. e.*, the patients are already themselves again, while the spasm still continues. This was very striking in one of my patients, where a rigid extension and stretching asunder of the fingers, that positively could not be overcome, lasted for some minutes after the brief unconsciousness had completely passed away.

Herpin remarks that in these forms consciousness is only slightly affected, not by any means wholly abolished. Although this may also occur, I must still, from my own observation, agree with Hasse that in the great majority an arrest of consciousness takes place, which, although often only very transient, is still complete.

D. *Irregular Forms of the Attacks, and the Epileptoid States.*

We speak of both together, because numerous transitions occur here, and a complete separation is not only contrary to the nature of the thing, but would also introduce much that is artificial into the description.

There is a great variety of forms under which at times the epileptic attacks may exhibit themselves, and which yet have long been recognized as such. They must be sketched first; we will afterwards return to the epileptoid states, properly speaking.

To-day it is regarded as almost an axiom that the most essential character of the seizure is to be sought for in the loss of consciousness. Now, there are, however, in contradistinction to all those hitherto described, certain *cases in which the coma is absent.* We do not mean simple vertigo epileptica, but paroxysms with distinct convulsions, but without arrest of consciousness. At all events, the spastic symptoms are here chiefly of a local character, analogous to those which we have described among the transition-forms (C), but there occur, although very seldom, strongly marked tonic and clonic epileptic twitchings, in which the patient, although all the time possessed of consciousness and sensation, at the utmost experiences a slight confusion and obscuration of his senses. I saw a pronounced case in a young fellow of sixteen, P. W.; in this instance typical paroxysms were observed, in which the patient's face at first became of a deathly pallor; he then lost consciousness and fell into general twitchings; these alternated with others, in which, without this pallor and without coma, violent clonic spasms appeared occasionally, but only in the arms and legs.

The literature of the subject contains a series of examples,[1] where at times the abnormal motor activity displayed itself in a different way from the tonic and clonic spasms heretofore described. Thus it has been observed that the patient at first ran violently, but was even then unconscious and at once began to suffer from the twitchings, or he began by moving round in a circle. Sometimes, also, *the convulsions are entirely wanting, and are only replaced by the motions of walking and running.* I have myself had a patient under treatment in whom, in place of the usually pronounced major paroxysms, there occurred at times some in which he ran up and down his room in a state of complete unconsciousness. Hammond relates an analogous case, and Trousseau another.

The greatest attention of physicians, especially in forensic medicine, has long been turned to the important cases in which epilepsy has declared itself by periodically recurring *attacks of mental disturbance* instead of the ordinary major or minor paroxysms. We shall see, hereafter, when we consider the immediate abnormal consequences of the ordinary attacks, that the severest states of excitement may arise in connection with an attack, just as we saw that mental changes may occur beforehand as prodroma. Thus it may also happen in a more extended sense that mental disturbances arise in place of the whole seizure, and present a picture completely at variance with the usual one of epilepsy.

For the details from a psychiatric and medico-legal point of view, we must refer to the special works treating of them. We can here take up only so many of them as necessarily belong to the outline of an exposition of epilepsy in general. This topic is treated of with considerable fulness by Falret,[2] and we follow him in essentials.

Falret believes that in the *delirium epilepticum* a slighter and a severer form must be distinguished, just as is done in the ordinary seizures, and that they present differences in the degree, but not in the essential nature of the symptoms. It is common to both forms for patients to be affected paroxysmally with an excitement which is at times announced by warnings, but often breaks out quite suddenly. This excitement, almost without exception, has a maniacal character, and is evidenced by the violence and suddenness of the acts which the patients perform. It disappears almost as rapidly and unexpectedly as it came, and the patient returns to his previous mental condition; he has afterwards either no recollection at all of the paroxysm and the acts done in it, or a very dim and confused one. There are various intermediate forms between the slight and the severest cases.

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[1] Compare the older ones cited in *Wicke's* Versuch einer Monographie des Veitstanzes.

[2] Archives génér. de méd. 1860. Vol. II. and 1861. Vols. I. and II.

In the mild form of epileptic delirium the inclination to wander round sets in, either after the previous occurrence of the mental prodroma which have been mentioned, or, sometimes, very suddenly; or the patients, as they afterwards assert, "are driven by an irresistible force" to perform acts of violence. Most frequently they throw themselves upon other persons and strike them, or they destroy objects about them, or they even wound themselves in a senseless way. At other times their acts are wanting in this character of excitement, and, instead, the most singular things make their appearance. Thus cases are known in which delicate and perfectly respectable persons suddenly use the most obscene language for a short time. The case reported by Trousseau is often quoted, where a high judicial officer suddenly, in the midst of the session, went into the adjoining room and there urinated in the corner, then returned to the hall and continued the session, and had no recollection of what had occurred. A patient under my observation was at times subject to a peculiar confusion, so that she committed the most foolish acts; *e. g.*, if she had just made a fire on the hearth she put into it knives, spoons, and whatever happened to be in her hand, instead of wood. After the lapse of a few seconds or half a minute, she came to herself again and was conscious of the acts of folly she had committed. The symptoms of paramount importance, however, and which are constantly referred to, are when in paroxysms of this kind patients are sometimes urged, by a sudden motive "impulse," to certain acts which ultimately assume a character of public danger. Here belong, at least in part, the violently contested questions of pyromania, kleptomania, and dipsomania. It is far from our intention to discuss this question in the present place; it belongs much more in the domain of the alienist and of forensic medicine. We would only remark, in accordance with the careful observations of various authors and the material, scanty, it is true, which we have ourselves seen in this connection, that it appears unquestionable that such a transient mental disturbance takes the place of an attack as an expression of epilepsy.

The severe form of epileptic delirium exhibits much more formidable features than the mild, for in this a transient, furious mania of the most violent kind breaks out, and is hardly surpassed in intensity by any other sort of maniacal aberration. Patients of this kind are met with almost exclusively in institutions, at any rate after the first seizure has occurred. We have indicated above, after Falret, the general characteristics of this delirium. Still we must here call special attention to the reckless impulsiveness of the acts, for they generally culminate in a senseless destructive fury. Patients annihilate everything about them; the most shocking murders, perpetrated in this way, are recorded in the annals of science. At times certain mental prodroma make the unhappy patient aware of the approach of the seizure, so that he can warn those around him to protect themselves (we ourselves know of a pronounced case of this kind); but at other times the passion for destruction breaks upon him so suddenly that no escape is possible. The sufferers often relate afterwards that they have had hallucinations, usually of a repulsive and frightful character, in which a red color has recurred in varied fashion with notable frequency. It also happens that patients are very violently but rather connectedly delirious.

After an average duration of from two to four days the maniacal condition passes off and the sufferers return to a rational state—provided, of course, their mental powers are not too much enfeebled—without retaining, in favorable cases, more than a dreamy consciousness of what has occurred. Sometimes this transition is accomplished in a few hours.

We now turn to the description of the *epileptoid states*. We have already pointed out in the introductory sketch that numerous wholly indeterminate morbid features were included under this name by Griesinger, but we have also mentioned the protest entered against it by Westphal. In the essay,[1] which he unfortunately left incomplete, Griesinger speaks especially of two groups of symptoms which he ascribes to epilepsy: the first group included many kinds of attacks of giddiness; the second, morbid states, which in practice are usually regarded as hypochondria or hysteria, but which, according to Griesinger, are epilepsy with strongly marked intermediary symptoms and very slight and incomplete seizures. Westphal, on the contrary, urges that such epileptoid seizures are among the most frequent symptoms in almost all the different diseases which belong among the mental and nervous affections. Taking this view of it, then, we should have to reckon general progressive paralysis, *e. g.*, as epilepsy on account of these epileptoid or epileptiform seizures. There is doubtless much that is correct in this objection of Westphal; but, on the other hand, we believe the same should be said of Griesinger's theory. In fact, so long as our knowledge of the nature of epilepsy is so inadequate, and so long as any one may, and even must, without contradiction look upon all the different groups of symptoms described above as epileptic seizures, it appears to us that we cannot deny the right of considering other combinations of symptoms also, under the circumstances, as the expression of epilepsy. If, now, we would not fall into the error so properly emphasized by Westphal, the boundaries must be clearly defined within which we are still to regard an anomalous combination of symptoms as an expression of epilepsy. In this view we would enunciate the following rules:

Those states should be regarded as epileptoid, *i. e.*, as caused by a central epileptic change, in which symptoms show themselves paroxysmally, for the development of which the same physiological processes, according to our present knowledge, should, or, at any rate, may be assumed, as produce (when developed in greater intensity or extent) the usual epileptic attacks. Furthermore, it should be kept clearly in mind that, as in genuine epilepsy, these paroxysms constitute the principal feature of the disease, while the symptoms of the interval, on the contrary, stand in the background, or are so little pronounced that no other nervous or mental affection could, with any probability, be made out from them. The only exception would be when they can be shown to be in some sort a consequence of the epilepsy. The *certainty* as to whether the case be one of genuine epilepsy or not, is to be gathered from the occurrence sooner or later of real paroxysms in place of, or alternating with, these questionable seizures.

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[1] Archiv f. Psychiatrie und Nervenkrankheiten. Vol. I.

We by no means fail to recognize the fact that even on this theory there may be discussion about many cases as to whether they should be classed as "epileptoid" or not. Still, the way of looking at it proposed by Griesinger appears to us, practically and scientifically, very important, and it was for this reason that we desired to make at least an attempt to formulate this question in more precise terms.

What now are the clinical features of these epileptoid states? It would be impossible to detail them here fully in their multiform variety; we can only give a characterization of a few types.

In the first series we must mention "*attacks of giddiness*," as well as the mere slight sense of oppression, without the actual loss of consciousness, as in epilepsia mitior. These vertigos are, as Griesinger properly insists, often ascribed to all possible causes, congestions of the brain, disturbances of digestion, and the like, while epilepsy is not thought of. The same investigator points out more fully what are the indications which should arouse our attention in such a vertigo, in a way to make our diagnosis positive. From the various histories of patients under my observation, I extract the following, as well describing this epileptic vertigo:

A clergyman, of twenty-eight, had frequently suffered as a boy from night terrors, which he called "nightmare." No hereditary influences are to be discovered. He was healthy up to two years ago, when he suddenly, without warning, became "faint," fell down, and was unconscious; afterwards, for three days, he was very weak and afflicted with headache. Since this time he has suffered, without having in any way changed his mode of life, from very frequently recurring attacks of vertigo. These are often preceded by palpitations of the heart; sometimes they are introduced by a peculiar "twisting" sensation in the region of the stomach, whence it rises "into the throat," and even up to the back of the head; then the patient's face becomes red, he has a sense of heat in the head, and is seized with such a decided giddiness that he has to take hold of something to avoid falling. Sometimes, in connection with it, a general trembling and qualmishness runs over him. These seizures come frequently, in part after mental exertion, but in part quite spontaneously, even when lying still.—It is now still further characteristic that the patient, after having latterly for almost two years been afflicted with these attacks of giddiness only, has had four attacks in which he fell to the ground, became very pale, according to the statement of those about him, lost consciousness completely for from one to two minutes, and was possessed by a general spasmodic rigidity.—Objectively there is not the slightest thing to be discovered.

If we analyze this case more thoroughly, the following circumstances present themselves which lead us to look with suspicion upon the vertigo: in youth, the striking nervous manifestations; an unmistakable aura proceeding from the stomach; marked change in the color of the face; palpitations of the heart, without the least change in the circulatory apparatus, and often without anything to occasion it, as when lying still; sometimes trembling at the same time. To these there were added, as the case went on, unmistakable epileptic paroxysms, with eclipsis and muscular rigidity.

The following condition, although presenting wholly different characters, must, I believe, be considered as epileptoid:

The patient, a robust man of thirty-two, is stated to have come from a healthy family; was formerly healthy; had only suffered while a student from "a slight sense of oppression in the head." In his youth he had been somewhat addicted to onanism. The present occurrences have existed for four years, and recur at intervals of from a week to a month, while between times the patient is perfectly well. Either spontaneously or after mental efforts there suddenly begins a glittering before the eyes, with obscuration of the visual field, especially on the right side. Then a peculiar sort of dreamy condition sets in; the patient recognizes his surroundings, but his thoughts are, "as it were, under a spell," and he has to think constantly of one and the same circumstance. He hears what is said by those around him in a dull way, and as if from a distance. This condition lasts for about half an hour, during which time, according to the statement of his family, his face assumes a very pallid hue. After the seizure there is a feeling of creeping and numbness in the right or left half of the body. Then he is again perfectly well.

Finally, we also present as an illustration the medical history of a student of theology, twenty-three years of age.

A brother suffers from pronounced epilepsy. The patient himself was entirely well up to his eighteenth year, only he masturbated between the ages of twelve and eighteen. Then one day, at dinner, without any known cause, he suddenly became dizzy and at once fell down unconscious; it cannot now be ascertained whether there were convulsions with this seizure. Since then seizures of the following kind have developed themselves at intervals of from one to three weeks, during which the patient has been perfectly well. Be the patient where he will, he suddenly experiences a peculiar impression of white light, not to be more accurately defined. Thereupon he believes that he is transported to some entirely strange, and usually imaginary region ("heaven or hell"), though he still knows that he is in the street or in college, and can even salute passers-by in this dreamy state—consciousness is never completely lost. His mother states that the patient during the seizures looks flushed, but afterwards pale, and that his hands are "spasmodically clutched together."—The patient made use of a cold-water-cure and of potassium bromide and ext. cannabis indica in large doses. The seizures described disappeared; but others set in, which last one or two minutes and in which the patient is actively excited, speaks confusedly, and is abusive.

We do not wish to add to the number of these examples, because it would be impossible to exhaust the great variety of the features they present. For our part we are of Griesinger's opinion, that such states belong to epilepsy. The aim of the inquiry is to establish where the limits of the territory must be drawn, and whether various other states should and must be included in it, of which we can take no account here in a manual, because their title to belong here can be still further contested, as in the case of the epileptoid neuralgia of Trousseau and others.

We now once more return, after describing the various forms of the paroxysms, to their *immediate consequences.* It was stated above that, as a rule, the patient returns to his normal condition after a deep sleep of longer or shorter duration; after the mild attacks, often with surprising quickness. Still at times the paroxysm comes to a close in another way.

In the first place it is to be stated here that very marked conditions of exaltation may follow upon the convulsions. Their character is generally perfectly analogous to that of epileptic delirium, *i. e.*, the patients are very highly excited and maniacal, and the delirium is characterized by the same senseless, impulsive outbreaks of fury as in the cases where it takes the place of the whole seizure. A further description may therefore be omitted. This mania lasts for a variable length of time, for one or a few hours, sometimes even for two or three days. On other occasions, the patient for a considerable time after the seizure is stupid, apathetic, and only very gradually comes back to his former status. Again, at other times, simple conditions of hallucination or somnambulism are established.—As a matter of experience, these serious consequences are almost exclusively observed in case of epilepsia gravior, although the intensity of the seizure and its immediate sequelæ by no means always stand in the same relation to each other, as it also happens that occasionally a milder epilepsy is followed by an uncommonly violent and protracted after-stage.

Certain other manifestations are also at times to be reckoned among the immediate consequences of the seizure, which, from their clinical features, we may characterize in general as *paralytic manifestations*, with which aphasia would also be associated. The paralytic manifestations are partly motor, partly sentient, and partly sensorial, such as deafness, loss of the sense of taste and of sight. Echeverria counts up a great plenty of them. Todd was the first to describe "epileptic hemiplegia" fully; recently Hughlings Jackson,[1] especially, has given close attention to the study of it. According to our experience, we must agree with Reynolds and H. Jackson that these resulting conditions or complications of the attacks are not directly dependent upon them as such.[2] With regard to the cases where a *lasting* paralysis remains after an epileptic, or in this case more properly an epileptiform, seizure, there can be no doubt that some grosser material change of structure exists within the skull, that in fact we can assume the existence of no epilepsy in the true sense of the word, but that the convulsions are simply the symptomatic expression of an anatomical cerebral lesion. Moreover, when these cases are investigated, further symptoms will be found without trouble, which are referable to such a lesion. Usually there are tumors, embolisms in the territory of the artery of the fossa of Sylvius, less frequently hemorrhages; still, in reference to the latter, we would add, so as to avoid misunderstanding, that we mean primary hemorrhages, such as are themselves followed by convulsions, not such as might originate during the seizure as a result of the venous stasis, because, as we have already remarked several times, this last accident hardly ever occurs. As we cannot enter more closely into this subject in this place, we must refer the reader to the other sections of the present work which treat of it.

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[1] Compare, besides the various articles in the journals, his article on "Convulsions" in *Reynolds'* System of Medicine.

[2] As *Reynolds* forcibly expresses it: "There seems to me to be no more reason for connecting the paralysis with the epilepsy than there would be for us to connect bronchitis, cancer, or corns with this disease."

We can express ourselves just as briefly with regard to the unilateral convulsions and the hemiplegia—permanent or transient—which follows them. It is true that sometimes unilateral twitchings occur even in genuine epilepsy, as was stated above under the head of irregular forms of the seizures; but these are almost always rather partial and insignificant, and at the utmost they are occasionally interchangeable with complete paroxysms —and even in these, if they recur with increased regularity, we must always be suspicious of an organic cerebral disease. If, however, a patient suffers exclusively from violent unilateral convulsions, and if transient or permanent paralyses or pareses of the affected parts remain after them, we may almost always with considerable certainty assume the existence of an organic brain lesion.

Now there are also cases in which patients suffer for a considerable time from completely developed epileptic paroxysms, which are frequently repeated, and after which there remain constantly, or, at all events, quite often, pareses or paralyses of motion or sensation, which are recovered from again, however, after some time. What is the state of things in these cases? Even if it be not possible to assign a cause with absolute certainty, still the conditions hitherto known to have been found on post-mortem examination and a full analysis of all the symptoms indicate that here also the grosser anatomical lesions always come into play. As in the case of epileptic hemiplegia, the site of these latter is often to be sought for on the convexity or in the hemispheres of the cerebrum. When speaking of the etiology, we have expressed ourselves to the effect that in our opinion such anatomical lesions of the cortex are capable of producing the "epileptic change;" in this way we could actually speak of an "epileptic hemiplegia" if a true epilepsy originated in this way, still this would be only a play upon words, for, in fact, it is not the attack which occasions paralysis, but the anatomical lesion itself. It is true, the method of the pathogenesis still remains obscure. We may, at most, conceive that transient disturbances of the circulation round about the anatomical lesion, which accompany or result from the attack, give rise to the transient paralysis.

Aphasia after epileptic attacks we only mention here by name, as this combination of symptoms is treated of in its proper connection in another part of the present work.

On beholding a severe seizure, it would be thought, especially by the laity, from the violence of the symptoms, that death would more often occur. On the contrary, it is, in fact, astonishing how very seldom the individual paroxysm as such causes death by the convulsive movements and their immediate consequences. At the utmost it is occasioned by unfortunate accompanying accidents, as, *e. g.*, by severe wounds, or most often in this way, viz., that the patients, if alone, fall so unluckily that they lie somehow upon the face, and the mouth and nose are obstructed, as a result of which suffocation of course follows ; or, again, by a seizure occurring just as they are eating, in which case the food gets into the larynx and may lead to immediate suffocation. Suffocation from spasm of the glottis, however, is among the greatest rarities. The case is different, of course, if the paroxysms are very frequent and follow rapidly one upon another ; then, it is true, patients not infrequently die, and in quite a variety of ways. We shall shortly recur to this more in detail.

Frequency of the Seizures.

Observations have always been made, of course, as to the frequency of the epileptic seizures, and among the more recent investigators, Reynolds, especially, has busied himself particularly with the inquiry into the question of how all sorts of influences affect their absolute frequency, their form, and their mutual relations. Detailed discussions of this sort, which are appropriate in a monograph, can only be touched upon in a manual in their most essential parts.

The prevailing rule is that the paroxysms generally recur very irregularly. The literature, it is true, furnishes the records of many instances in which a certain periodicity, a certain rhythm is not to be denied, and every physician of some experience will be able to add to them. Thus, in many patients the paroxysms recur at definite times of day, or once a week, or once every two weeks, or every other day; sometimes with women, in a certain loose connection with menstruation, or, at all events, monthly, from which has arisen the wholly untenable popular belief in the influence of particular phases of the moon. For all these cases it holds true that such a rhythm is only temporary, transitory, frequently even only apparent, because quite inconstant, and that with the same patients the seizures may again depart widely from the type. The rule, then, is the irregular recurrence, and more or less regularity is to be regarded as an accidental circumstance, not depending upon the nature of the disease or upon regularly recurring extraneous influences.

The frequency of the paroxysms varies within extremely wide limits; some patients have one seizure a year, others have thousands. We regard it as rather an unprofitable piece of trouble to count up the numbers so as to get at the average frequency in many epileptics. Even in one and the same patient, however, a noteworthy variation sometimes takes place, although, when calculating for considerable periods, the average with the same individual undergoes slighter changes; thus there may be one paroxysm weekly, then after one weekly interval there may be three or four, then again a cessation for two months may ensue, and then again a seizure. The free intervals may become still greater, even apart from the influence of any medication, a circumstance which must be recognized and taken into consideration when estimating the value of therapeutic measures. After such a considerable pause the first paroxysms which again occur are apt to be of greater violence, and to appear rapidly one after another. Investigations as to whether the attacks occur more frequently at the beginning of the disease or increase in frequency as the duration of the malady increases, furnish no positive results. At times they are more frequent at the beginning and then diminish, and the opposite; only it might be said to be very generally true that when the epilepsy is incurable, as its duration increases, the frequency of the paroxysms often increases.

Besides, no definite numerical statements can be made as to the proportionate frequency in the same patient of isolated attacks and those which occur in groups. These groups of seizures themselves, however, require a more minute discussion. They may be composed of from four to one hundred single seizures, or even more, in twenty-four hours. Delasiauve saw in a boy of fifteen within *one* month a "collective seizure," which was composed of the incredible number of 2,500 "fragmentary seizures." It is evident that so numerous attacks rapidly succeeding one another must have an extraordinary effect upon the patient. The French have introduced a special name for this state in which the patients remain in a coma in consequence of the rapidly following attacks, viz., *état de mal épileptique* (status epilepticus—compare the note given on p. 188). Bourneville (loc. cit.) gives detailed attention to this very subject; Obersteiner,[1] too, has quite recently described three cases of the kind, and we have ourselves once observed a case of the same sort. In Bourneville's account an enormous increase of temperature, which may even rise to 42° C. (107.6° F.), is brought forward as specially characteristic. Starting from this period, the patients lying in a deep coma and with the temperature mentioned, a turn may be taken in several directions. Either the temperature advances still further and a fatal result takes place, or, on the other hand, all the symptoms gradually subsiding, the patient recovers;—the former event seems to be somewhat the more frequent. At other times, even if death be not immediately dependent upon the convulsions, the case may still take an unfavorable issue; the temperature advances anew, consciousness becomes still more obscured, and the patient succumbs, sometimes being afflicted with bed-sores, with the symptoms of profound collapse. At times even this second advance of temperature is overcome.—The post-mortem appearances, as brought forward by Bourneville, give no satisfactory explanation of the cause of death. In two cases under my observation, after seizures thus frequently repeated, there set in marked œdema of the lungs, in one of them a commencing pneumonia also, and the patients died with the symptoms of these affections in deep stupor and high fever. Westphal also witnessed an advance of temperature with paroxysms following *coup sur coup* and ending fatally. It cannot at this time be positively decided whether this high temperature in the *état de mal* depends simply upon the violent convulsions, or whether it is to be set down as analogous in its own peculiar nature to the as yet unexplained rise of temperature in the death agony in diseases of the central nervous system. It appears to be unquestionable that the latter point also bears its part in the cases that end fatally; still, we cannot well speak of a rise of temperature in the death agony when the patient recovers, and in those cases the effect of the intense muscular movements must be brought in to assist in explaining it.

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[1] Wiener med. Wochenschrift. 1873. 23.

Bourneville observed hemiplegia in the majority of the cases that he saw. Those where an autopsy was made confirmed what was stated above incidentally to epileptic hemiplegia, viz., that gross anatomico-pathological conditions were found. In no instance, however, is this hemiplegia in any way characteristic of the *état de mal.*

The fact that the seizures often appear *at night*, during sleep, is of great importance. In regard to this, too, statistical inquiries have been undertaken, from which we only arrive at rather indefinite results, for it has been ascertained from a very large total, that on the average "epilepsia diurna" is somewhat more frequent than "epilepsia nocturna,"—so far at least as information regarding night attacks could be obtained. Of course the same patient may occasionally have his attacks just as well by day as by night.—Trousseau groups together, in a practical way well suited to the purpose, the conditions in the presence of which we may, with more or less certainty, assume that seizures take place at night. This is important to know, because it happens that with some patients epilepsy begins with attacks exclusively nocturnal. Our attention will be attracted by the resulting mental conditions spoken of above, such as depression, stupidity, headache, and the like, if persons previously healthy complain of these on some occasion on waking in the morning. We attain to certainty if involuntary discharges of urine or fæces have taken place in the night, if the tongue has been bitten, or small hemorrhages into the skin are found.

We have so far spoken only of the frequency of the seizures in general; *the relative frequency of the various forms* now demands a brief consideration. The results arrived at by various observers in regard to this differ from each other in some points, but agree in others. Except where we make some special comment to the contrary, we can confirm, from our own experience, the following propositions, which were in part established by Reynolds:

There are epileptics in whom only the major attacks take place, others again who suffer from the mild form only, with no pronounced spasmodic manifestations, or but very few. The latter group is very small in proportion to the whole number of epileptics, the former very much larger. There is a general agreement so far as this. Not so with reference to another question. There are, namely, in the third place, patients in whom, as we have already pointed out incidentally, the different forms of the seizures occur jointly, and they consequently have alternately major attacks and mere fits of dizziness, with or without partial twitchings. It is confirmed by all that this combined form is more frequent than the epilepsia mitior alone; it is only a matter of discussion whether it is rarer than or nearly as frequent as the pure epilepsia gravior. From my own experience, I adhere to the opinion that the combined and the uncomplicated severe forms are just about equally frequent. The influence exercised by the combination upon the course of the disease we shall touch upon further when speaking of the prognosis. According to Reynolds's careful inquiries, we ought not to take the view that the number of the major seizures would be diminished by intercurrent light ones, or that again the latter occurred vicariously for the former, but that the *petit mal* is a simple advance in the intensity of the disease.

Epilepsy may first manifest itself in the form of either the greater or the lesser attacks. Reynolds has established the fact that where the tendency is inherited, the onset is more commonly with the major than with the minor. Echeverria confirms this. In determining the character of the seizures, the age at the beginning of the disease is of much less essential significance. It is just as little susceptible of proof that the length of time during which the malady has lasted exercises any modifying influence upon the nature of the attacks

II. *The Interparoxysmal Condition.*

Although the seizures constitute the peculiar essential symptoms of epilepsy, still the condition which exists during the interval between them also deserves attention corresponding to the often strongly marked symptoms which it offers. This attention has already been given to it in part, particularly with reference to the mental state of the patients. Its importance, however, was first fully appreciated by Russell Reynolds, at whose hands the subject has received a careful and thorough examination.

General Constitutional Relations.

A decided protest against the opinion which has prevailed since Hippocrates, viz., that epilepsy may occur, on occasion, in any state of the constitution, has been offered almost alone by Radcliffe, and he maintains that the peculiar, typical condition of the system at large in epileptics is weakness and depression of the circulation (the powers of the circulation are always defective). This view of Radcliffe's, however, has not been assented to, because it is too much opposed to every-day observation. There are undoubtedly miserable, weak epileptics enough, but those are decidedly more numerous in whom the bony framework, the muscular strength, the color of the skin, the deposit of fat and the energy of the condition of the circulation do not depart in the least from the normal; there are even occasionally some who are noticeable for their Herculean frame and strength. It is equally impossible to prove that the duration of the disease leads to any habitual changes in this respect.

As the constitution may be perfectly normal, so in the case of certain epileptics may *all* pathological appearances on the part of the nervous system be absent; *i. e.*, many epileptics appear to be ailing only at the time of the paroxysms, exhibiting in the intervals *the appearance of thoroughly and completely healthy persons.* And in fact this obtains not merely with recent patients, and where the paroxysms are infrequent, but even sometimes where the duration of the disease has been considerable, and after frequent seizures. This fact is of high significance, for it proves, first, that the seizures are actually the essential feature in the disease, because the constant one; secondly, that the symptoms of the interval, because inconstant, may depend upon merely accidental or secondary changes; finally, that we are right in seeking for the primary and proper seat of the epileptic change in the medulla oblongata and pons, and not in the nervous system at large, precisely because, as we shall prove further on, the manifestations of the seizure may be derived from antecedent processes in these parts alone, while the symptoms of the interval, which must be referred to the cerebrum and ultimately to the spinal cord, may be entirely absent in the disease when fully developed.

The interparoxysmal manifestations which occur may involve the motility, the sensibility, or the mental faculties.

Motor manifestations.—When speaking of the motorial aura it was stated that the spasmodic movements of which this aura consists were also often observed without a seizure following. But associated with these there also occur motor phenomena in the intervals. As rightly observed by Reynolds, and as can be easily determined, they may show themselves in a threefold form: as a simple muscular trembling, as clonic, or as tonic spasms in single groups of muscles. Quite frequently we have two or even all three of them together. The clonic spasm is the most frequent, the tonic the least so. I have seen a perfectly typical instance of the latter, as the only symptom of the interval, in the shape of a periodically recurring cramp of the calf. According to Reynolds, some seventy-five per cent. of epileptics exhibit some such motor change in the intervals.

Sensory manifestations.—These are rather less than more frequent than the motor manifestations. Quite apart from the fact that the disturbances exhibited by the sensory aura also occasionally occur alone, without any subsequent seizure, that they are, in fact, symptoms of the interval, we chiefly observe headache, persistent sensations of dizziness and dull confusion of the head. It is only exceptionally that these attain an especially high degree.

Mental manifestations.—The fact that disturbances of the mind are present during the intervals has been known for a long time, and has always commanded the greatest interest. Many alienists have even gone so far as to declare such epileptics insane. This opinion is, however, decidedly incorrect; it can only be a deduction from observations which have been made exclusively in institutions. The results of those which have been collected from private practice are of a wholly different tenor.

It is undoubtedly possible for an absolutely healthy state of mind to coexist with epilepsy. Reynolds, in fact, reckons from his own cases that this is true of a third of all patients. It is well known that historical tradition tells of numerous very highly gifted men who suffered from epilepsy, and whose deeds do not allow the recognition of any mental deterioration (Cæsar, Napoleon, Petrarch, and others).

If mental disturbances occur, as is certainly the case in the majority, according to what has just been said, these may again be very various in their nature, and especially in their degree. There are all transitional stages from such slight alterations that they can be discovered only by careful attention, up to the highest degrees of mental disease, in which the human characteristics of the individual gain expression only in certain traits, perhaps only in the power of speech. We are not willing to reproduce such statistical statements as are available, because, although so far carefully compiled, they are still too small. Only be it remarked in general, that fortunately the slighter degrees are far the more frequent.

The most common and often also the first mental lesion is a weakening of the memory, and on this point we are agreed with others. Here again, as in almost all cases, the peculiarity obtains that the memory is principally enfeebled with regard to what has happened most recently, while it exists in perfect freshness with regard to things long past. A diminution in the sharpness of apprehension is decidedly less frequent. Then again, the character and disposition are frequently altered. Patients become gloomy, out of humor, depressed, or violent, irritable, "nervous," distrustful, easily angered. Or the disposition is changeable, often without any appreciable reason.

These slighter degrees of mental disturbance may now last for a year, or continue always to exist as such. At other times, however, a constantly advancing disease of the mental life then forms a transition stage to what is ultimately mental death or complete imbecility. Patients decline into marked hypochondria or melancholy. And further, to make use of the words of Griesinger, " the memory decreases, the imagination grows dull, the fancy loses its richness of coloring, its intensity and warmth, and the spirit is withered. The physiognomy and bearing are altered, the lips of patients grow thick, their features coarse, and they acquire a hideous expression of countenance. . . . A final decline into imbecility is the sad destiny of many of these patients. . . . Cases of epileptic imbecility are to be regarded as wholly incurable ; the other forms of insanity, complicated with epilepsy, as curable only in exceptional instances."—Further details in reference to this subject belong in the department of mental disease, and therefore cannot be touched upon here.

On the other hand, we now turn to a brief discussion of a very important question, which, again, has been most carefully treated in its various aspects by Reynolds, whose statistical results form the basis of what follows. Are there any definite conditions, which, belonging to the nature and progress of epilepsy, have an action which favors the development of mental lesions?

Hereditary predisposition and the time of life at which the disease began, as well as general constitutional relations, play no appreciable part in developing them. According to Reynolds, however, the length of time during which the epilepsy has existed has just as little significance. This view, which is opposed to that advocated by Esquirol and many others, evidently finds its support in the simple fact that there are many persons advanced in life, who have been epileptic from their youth, and in whom there exist only slight mental disturbances or none at all. But a short time ago I treated a lady who had been epileptic for twenty-six years, and who suffered from only a moderate impairment of memory and at times from a somewhat sad disposition, and in whom the power of judgment was only slightly impaired.

There is a prevailing agreement in the opinion *that no relations of any kind whatever exist between the mental disturbances and the seizures.* Here Reynolds, by an exact statistical method, has arrived at noteworthy results which differ in several respects from the generally received opinions.

The first result arrived at is that the number of the paroxysms simply, through which the patient has passed, stands in no relation whatever to the mental change; how large soever the number may have been, by that circumstance alone the intellectual faculties are not impaired. Reynolds asserts that eleven men under his observation had endured an average of 2,000 seizures (one 18,455, seven more than a thousand) without a single mental disturbance having followed. On the other side, such a change has been observed after no more than from six to twenty paroxysms.—Moreover, the opinion that the more violent the separate attacks are in their symptoms, the more the mind suffers, is not confirmed by close examination.

On the other hand, it appears that the rapidity in the reappearance of the separate attacks is of a greater significance, in that the intelligence is more impaired by a frequent recurrence of them. Yet even here there is no constant ratio of dependence, for now and again certain patients remain free notwithstanding that the seizures return often, and, on the contrary, others have their minds affected even with infrequent attacks. This latter I have myself seen in one case where there was a repetition of the attacks but four times a year; it is true, the lesion was a very slight one, so far as my observation of this patient extended.—As regards the form of the seizure, it may still be stated that many persons regard those cases as decidedly the more unfavorable, so far as the development of intellectual disturbances is concerned, in which epilepsia mitior is present together with the severer form of the disease.

We will now submit to a brief consideration the question *whether in any way single cases of epilepsy present clinical varieties in accordance with etiological influences.* The analysis of the individual cases of illness certainly teaches that this is the case to only a limited degree. Still we regard even a slight influence in this respect as important enough to be considered here in view of the significance of the question raised in its relation to therapeutics.

First, is epilepsy stamped with a special character by reason of an *inherited predisposition?* It is true, we have incidentally touched upon this point in the course of our exposition, but we wish once more to gather together here what there is that bears upon it. According to experience such patients sometimes suffer from eclamptic attacks even in their first dentition; if not then, the first beginning of the disease almost invariably shows itself in early youth, even up to fifteen or seventeen years of age, and only very rarely after twenty. Reynolds has concluded from his cases that hereditary predisposition is not without influence upon the character of the seizure, since when it exists epilepsia gravior is much more often developed without the milder form. Still the relation is by no means constant, for in certain cases epilepsia mitior also occurs. More important, particularly for prognosis, is the knowledge of the fact that, in spite of inherited predisposition to the disease, mental disturbances do not appear more frequently than where this is absent, while *a priori* one is perhaps inclined to make a more unfavorable prognosis in these cases.

Secondary epilepsy, i. e., that form in which peripheral or central anatomical lesions serve as the starting-point for the development of the disease, presents some peculiarities.

If a pathological condition of irritation of a *peripheral nerve* is the influence which gives rise to the epilepsy, its picture has the following special features: Almost invariably a certain length of time passes prior to the occurrence of the first seizure; at least it is so recorded in the observed cases which can with positive certainty be counted in this category. This is of variable length, averaging some weeks, but at times amounting to months or even longer. Even during this period painful sensations not seldom show themselves: either clonic or tonic spasms in the tract of the affected nerve, or the two combined. These abnormal phenomena remain confined to the nerves or they spread more widely, but scarcely ever extend beyond the side of the body originally affected. Then the pronounced spasmodic seizures appear with loss of consciousness. These are always ushered in by an aura, which may be in different cases of a motor, sensory, or vaso-motor kind, but, as a rule, is characterized by the fact that it is always of like nature in the same patient. It begins in the region of the injured nerve, and hence arises the importance of a constantly uniform aura in judging of a special case of epilepsy. If such be found, one must always be closely on the watch for some sort of a peripheral lesion. Not as if analogous uniform auras did not occasionally present themselves in other forms, but in those mentioned they are the rule. The symptoms above indicated often appear in the intervals, and they also precede the outbreak of the first attack.—Moreover, in these cases, if there happen to be a cicatrix on the body, one may sometimes, but not always, produce a seizure by pressing upon or touching it. At the same time it is worthy of note that an actual "epileptogenous" zone, as in the case of the guinea-pigs which were experimented on, is never distinctly marked. I have myself continually sought for it in vain in patients affected. So much the more interesting is the case of Ogle's, in which there was an extensive portion of the body, the upper extremity, by touching which a seizure could be produced.[1]

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[1] Lancet.

Somewhat different features, again, are presented by those epilepsies which owe their development to the existence of a small circumscribed *lesion of the brain* on one side. We must think of this form, if the following symptoms are present: the attacks begin either very suddenly—this, to be sure, would be without any significance; or, as is more frequently the case, they are introduced in some constantly uniform and peculiar way. There first occurs, for instance, a tonic or clonic spasm in a particular group of muscles or even only in a single muscle, as in the region supplied by the facial or even only by single branches of it, as in the orbicularis palpebrarum, or in the region supplied by the spinal accessory, or in certain fingers, etc. Hence the spasm sometimes spreads over the corresponding half of the body before it becomes general. I believe that great importance should be attributed to this peculiar stereotyped commencement. Here, on the other hand, the sensory aura, which plays so great a part in epilepsy from peripheral causes, retires decidedly to the background; it occurs only very exceptionally, if at all. Further than this, the occurrence of a paralysis or paresis, unilateral, or even limited to the tract of a single nerve, is of the highest significance, if it be present, though it is not constantly observed. We have already expressed ourselves more fully on this point above. We believe that no special weight should be attached to the other symptoms. In particular, marked and characteristic symptoms in the intervals would give the case the stamp of a symptomatic epilepsy, *i. e.*, one not strictly genuine.

Pathology.

In the introduction we have already expressed our opinion to the effect that we very decidedly consider epilepsy as a definite disease, well characterized cases of which are perfectly typical, and can be confounded with no other nervous affection. Then, to be sure, as we depart from this fixed centre, cases at the most various extremes occasionally come under observation, in which it may well be a matter of discussion whether they still belong to epilepsy, so indefinite are the features of the picture. Still, epilepsy has this peculiarity in common with many other affections, even such as are well marked and are so-called anatomical organic diseases.

Another point, certainly, is much more interesting and important, and that is *the numerous connections* which *epilepsy has with various other nervous affections*, the intimate reciprocal relation in which it stands to them. Naturally this obtains reciprocally for the latter just as much, but the relation in the case of epilepsy is more striking, because this is one of the more frequent among the well-characterized nervous affections.

Now, these mutual relations are variously exhibited. In the first place, they are manifested in the matter of hereditary transmission. Concerning this we refer to what we have said above when speaking of the etiology, and do not consider it necessary to refer to it here again. Then again, in one and the same individual, epilepsy may exist side by side with other nervous diseases. We have already mentioned the mental disturbances, when speaking of symptomatology; but other neuroses may also be present at the same time with epilepsy; thus, *e. g.*, the combination with hysteria is by no means extremely rare;[1] we have ourselves seen chorea minor and epilepsy at one time in the same patient. Moreover, in an individual afflicted with a hereditary neuropathic condition, several forms of nervous disease may appear one after another; in childhood St. Vitus's dance, later epilepsy, or else catalepsy, migraine, and other neuralgias, form the advance-guard of epilepsy. Furthermore, such cases as the following are in the highest degree instructive, *e. g.*, two which have recently been published by Hitzig,[2] where after an external peripheral wound there first developed in the same patient epilepsy and then choreic movements, and in a second paralysis agitans, and then epileptoid seizures.—Of course, we cannot here enumerate all the various possibilities which have been noted in individual instances; we will refer to only one very interesting relation. There are namely a number of cases where certain members of neuropathic families have suffered from diabetes mellitus, while the others were mentally diseased, epileptic, or generally "nervous;" also saccharine urine and epilepsy have been observed in one and the same patient simultaneously.[1] Ebstein reports the same thing with regard to diabetes insipidus.[2]

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[1] Of course, we are not speaking here of the seizures of so-called hystero-epilepsy.

[2] Untersuchungen über das Gehirn. Berlin, 1874. p. 186 et seq.

In view of our almost complete ignorance of the real nature of almost all of the diseases indicated, it would be a mere idle playing with words if we should attempt to discuss these reciprocal relations more minutely. This must be postponed to a future time when we are better informed; for the present we must be contented with simply placing the facts on record.

It may be regarded almost as a necessary postulate, speaking from the present standpoint of science, that a disease of such long duration and with a group of symptoms in most cases so distinctly marked, must have as a foundation anatomical changes in some portions of the nervous system. But what are the parts *where the seat of epilepsy is to be sought?* Is it in definite regions, or spread more generally over the nervous system?

To arrive at a correct answer to this question one ought, in our opinion, to base the inquiry on only the really typical cases. If this be neglected, our position will be so far set back. As in the study of the malarial affections one would begin not with masked fevers, or such as are accompanied by severe cerebral symptoms and pigmentary embolisms, but with the ordinary forms, so in epilepsy should we follow the same course.

Now we have already stated several times that in determining the character of our disease, the real emphasis should be laid upon the paroxysms, for the very simple reason that quite often no symptoms whatever are present during the intervals. If, however, the seizures are the principal events, often alone betraying the epilepsy, then it can only be logical for us to locate the real seat of the disease in those parts of the nervous system from which the paroxysms take their origin. We shall have to prove hereafter that this part embraces the pons and medulla oblongata. *The pons and medulla oblongata are then the seat of epilepsy;* if any anatomical changes exist, they are to be sought for in these parts. Now, although up to the present time, as has been observed in the section treating of this subject, even histological inquiries have produced but a minimum of results, still even the little that is known about this is likewise calculated to turn our attention to the parts that are already known.

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[1] Compare *Griesinger*, Studien über Diabetes. Arch. f. physiolog. Heilkde. 1859, and Vortrag zur Eröffnung der Klinik u. s. w., ibid. 1866.

[2] Deutsches Archiv f. klin. Med. Vol. XI. p. 344 et seq.

The opinion just advanced as to the seat of epilepsy is shared by the majority of painstaking investigators of this disease; we mention, *e. g.*, only Schroeder van der Kolk, Reynolds, Echeverria, in part also Brown-Séquard and Kussmaul.

An attempt to generalize the anatomical seat of epilepsy has sprung up again in certain quarters, to extend it as much as possible over the cerebrum and spinal cord, just as the ancient authors prior to Bouchet and Cazanvililh already located it in quite a general sense in the brain. Still, in our opinion, the reasons now advanced in support of this are just as little conclusive as the earlier ones were. Thus they refer to the settled fact, which was also recently determined experimentally by Hitzig (*vid. sup.*), that epilepsy can be excited by wounding the cerebrum, in pretended proof that the seat of epilepsy must be sought in the cerebrum also. Hitzig himself has indeed been cautious about drawing this inference; he only prudently expresses the opinion that wounding the cortex of the brain may lead to epilepsy. We share this latter opinion entirely, as we have already declared when speaking of the etiology. Here we have to deal with exactly the same relation as in the case of peripheral wounds which are followed by epilepsy. In this case no one thinks of seeking for the seat of the disease in the wounded sciatic, nor in a case of tetanus do we look for the seat of the affection in the irritated branch of the plantar nerve, but we only fix upon the wounded peripheral nerve as the starting-point, and we must proceed in just the same way, in the case before us, with the lesions of the cerebrum.[1]—Just as little can we acknowledge another reason sometimes assigned for the "diffuse" nature of epilepsy. The cases with abnormal paroxysms, namely, are appealed to,—those in which mental disturbances are especially prominent, or where such actually take the place of the epileptic seizure (délire épileptique, etc.). We shall presently explain what interpretation, in our opinion, should be put upon some of these cases. For the explanation of others among them, however, it is amply sufficient to assume the existence of those physiological processes which are called into play in the ordinary attacks; and the departures from the usual form of the paroxysm are explained by merely unessential modifications of the processes present in all seizures. We shall discuss this point also by and by.—Finally, we may adduce as proof that there is no participation on the part of the spinal cord in the real epileptic change, and that the spinal cord is not to be regarded as the seat of epilepsy, the interesting experiment of Brown-Séquard's mentioned on a preceding page (p. 198), in addition to Kussmaul's and my own experiments with reference to its non-participation in the causation of convulsions.

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[1] It seems to us so self-evident that we should not conclude (as Ferrier, *e. g.*, does) that the seat of epilepsy is to be sought for in the cortex cerebri, from the circumstance of epileptiform twitchings occurring on active electrical excitation of the cor-

Now, although in regard to the majority, especially of typical cases, we firmly maintain that the real seat of the disease is in the pons and medulla oblongata, we wish to protest against the idea that we deny a participation of other parts also of the nervous system. Such a participation may happen in two ways. In the first place, it is possible that secondary changes may be set up, *e. g.*, as a consequence of the seizures, and caused by the processes to which these give rise, although we still have very little knowledge of these secondary manifestations. Perhaps dilatations of the vessels, *e. g.*, in various parts of the brain, or, again, lesions of nutrition of the nervous tissues, with their consequences, which are dependent upon anomalies in the circulation, are to be taken into account here. But then we must undoubtedly assume for a definite group of cases a more generally diseased condition of the nervous system, which did

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tex, that we do not believe it necessary to waste a word of proof on it. With equally good or still better reason have I been able to draw the same conclusion from my own experiments, in which a simple wound with a needle in the cortex cerebri produced epileptic seizures (comp. Virchow's Arch. Vol. LVIII.).

not arise secondarily, but which is primary equally with the epileptic change in the pons and medulla oblongata; this is especially so of cases where the epilepsy is not pure, but is combined with other neurotic and mental affections, independently of whether the latter preceded it or were present at the same time. Then, however, we have no longer exactly a pure epilepsy, and it would be self-evident that such cases could not determine the seat of typical epilepsy.

We now turn to the principal question, *In what does the essence of epilepsy consist? What are the morphological or functional changes underlying it?* The answer to this must unfortunately, even at the present day, prove very inadequate.

In the section on pathological anatomy we have seen that the gross anatomical and the histological discoveries so far made, on the one hand, refer to changes which are palpably secondary, and, on the other, are of the most varied character. No single, definite, invariably recurring abnormality of tissue has yet been determined. In so far as it is at present allowable and possible to arrive at a more general conception, I may express my views on this subject as follows:

Probably no uniform, constantly recurring histological change generally forms the basis of epilepsy. I am much more disposed to believe that various anatomical changes may call forth the group of symptoms which constitute the disease, provided that these changes always affect the same parts of the pons and medulla oblongata, *i. e.*, such as are anatomically and physiologically equivalent. The sensory nerve, for example, always responds with the same functional expression, *i. e.*, with pain, to wholly different impressions—in part those which are coarsely demonstrable, such as moderate compression or neuritis, in part those which elude anatomical recognition, such as malarial infection; the phenomenon of tremor, also, is excited by various impressions upon the spinal cord; and in this same way are we inclined to represent to ourselves the relation of the anatomical changes, whether coarser or finer, to the production of the epileptic group of symptoms. Further inquiry will teach us whether this view is true or false.

With regard to the other question—*What are then the func-*

tional changes which the pons and medulla oblongata undergo in epilepsy?—the answer can be given with somewhat greater precision. Schroeder van der Kolk, Brown-Séquard, and Reynolds have already expressed themselves to the effect that we have to deal with an "*increased irritability*" of the reflex nervous centres situated in these sections,—an opinion in which we thoroughly share. Or, to state the case differently: In consequence of the changes which, in epilepsy, occur in those ganglion-cells of the medulla and pons, through the agency of which reflex processes take place,—changes which at times are probably only molecular in their nature,—these reflex centres respond from time to time much more easily than normal ones to irritations which either act visibly from without, or are developed in some unknown way in themselves. At the same time these centres respond with motor manifestations the intensity of which far surpasses the normal degree, but which in their nature do not differ from the normal reflex movements.—We are certainly still wholly in the dark as to the influences which induce this exaggerated excitability. It is possible, in analogy with other physiological processes, that a plethora of blood in the parts of the brain concerned may play a part here, but it has not as yet been proved to be so. It is possible also, in regard to other cases, that, if the ganglionic apparatus and the conducting channels leading to them were thrown into activity at the time of an attack, and were affected by a violent form of excitation the first time, such as terror or some decided irritation coming from the periphery or the cortex, this apparatus would thereby get into a condition of unstable equilibrium, so as subsequently to respond to slighter irritations with an excessive reaction; and this is precisely what is exhibited under the form of the seizure Still, as all these questions and ideas are as yet merely hypothetical, we must here, in a manual, content ourselves with merely indicating them.

With the circumstance last mentioned, we have approached another point in the pathology of our disease which has been up to the present time wholly uninvestigated, and which, consequently, we can only touch upon cursorily. It concerns the question, *In what way do the different etiological influences*

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produce epilepsy? As to most of them, no answer whatever can be given; as to some among them, such as violent mental impressions, or decided peripheral irritations acting but a single time, we have already attempted to give an answer. But even for those cases in which the state of things is apparently the most transparent, any actual clue, in the shape of facts, is wanting. We are thinking, namely, of the secondary epilepsies which are excited by wounds of the cortex, of the spinal cord, especially in the experiment on guinea-pigs, and of the peripheral nerves. We are acquainted neither with the centripetal nor with the centrifugal channels along which the irritation is propagated, nor do we know of what kind the change so propagated is—neuritis, or what—nor even whether this dissemination takes place continuously or *per saltum*, although the latter is highly probable.[1]

Thanks to experimental investigations, we are better informed on the question of *the starting-point and mechanism of the epileptic seizure* than on the uncertain topics which have just been spoken of. In this direction the work of Kussmaul, especially, has paved the way. As we have already referred in general, under the section on "Experimental Researches," to the special investigations which here come under consideration (*vide supra*), we may limit ourselves in this place to a brief comprehensive statement.[2]

Two symptoms are to be regarded as *essential* in the seizure: first, the mental disturbance, which generally manifests itself as a more or less distinctly marked loss of consciousness; second, motor disturbances, under the form of more or less extensive convulsions. The *primary* starting-point for both is to be sought in the pons and medulla oblongata.

It certainly requires no discussion to show that the loss of consciousness depends directly upon an abolition of the activity of the cortex cerebri; but, in the first place, this participation of the hemispheres is secondary, while the processes which lead to it arise primarily from the medulla oblongata. Besides, there can be no doubt that not merely the nerve-fibres, but the ganglion-cells of the pons and medulla oblongata acquire activity at the time of the seizure. If a special proof were still required, this would be furnished by the peculiar form in which the convulsions are generally manifested in the seizure, to wit: as a rule, first a tetanus of brief duration, and then a decided clonic spasm. This form of movement can only take place when the excitation traverses ganglion-cells, with the exception of certain special conditions, which do not come under consideration when speaking of epilepsy.[1] The ganglion-cells, however, the excitation of which results in the general epileptic spasms, with participation of almost the whole muscular system, lie united in the parts of the brain above-named, as anatomical investigation and physiological experiment teach us. In the pons lies the "convulsion centre," *i. e.*, that tract, through the excitation of which general convulsions arise (compare above, p. 198). Deiters has pointed out that in the pons exists the first *central* termination of the motor-fibres which come from the periphery; at the floor of the fourth ventricle lie united the gray nuclei for the motor cerebral nerves; moreover, in the medulla oblongata is found the so-called respiratory centre, and finally also the vaso-motor centre, which, as we shall presently see, is concerned in the genesis of the cerebral symptoms.—We have already pointed out that epileptic convulsions do not originate in the spinal cord.

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[1] Compare *R. Klemm*, Ueber Neuritis Migrans. Inaug. Dissert. Strassburg, 1874.

[2] Compare my lecture, "Ueber den epileptischen Anfall," in Volkmann's collection of clinical lectures, No. 15, where I have made a brief analysis of its symptoms.

Not merely the convulsions, but also the other phenomena of the seizure, particularly the loss of consciousness, are explained by an excitation of the central apparatus located in the pons and medulla oblongata, and, in fact, of the vaso-motor centre. The symptoms so evoked by the activity of the nerves which constrict the vessels are in part accessible to direct observation, and we have learned to recognize them in the account of the way the seizure begins—with pallor of the face, dilatation of the pupils, and frequent arrest of the pulse. Now, what takes place in the vascular region of that part of the head which is visible obtains also within the cranium; anæmia occurs there also. At the present time this is to be regarded as mere conjecture, based only upon the analogy drawn from the anatomical relations. This assumption, however, has gained very essential support from what has been discovered with the ophthalmoscope, and from Brown-Séquard's observations on epileptic guinea-pigs.[1]

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[1] Compare *Setschenow*, Ueber d. elektr. u. chem. Reizung d. sens. Rückenmarksnerven d. Frosches. Graz, 1868, and *Nothnagel*, Zur Lehre vom klonischen Krampf. Virchow's Arch. Vol. XLIX.

That anæmia of the cerebrum can produce loss of consciousness we know directly from Kussmaul's experiments. In every respect is the conclusion justified that the epileptic coma is caused by an excitation of the vaso-motor centre (in particular, that for the nerves of the cerebral vessels) in the medulla oblongata.

According to my conception then—for the more particular elucidation of which I must refer to the lecture already cited—the convulsions do not depend upon an anæmia of the pons, acting as an excitant upon the convulsion centre, an opinion we sometimes find advanced; for, according to that, the primary and indispensably necessary element in the seizure would always have to be the excitation of the vaso-motor centre, and the effect of it—the anæmia—would be secondarily to throw the rest of the motor apparatus into activity.—That anæmia of the pons *can* occasion convulsions is placed beyond all doubt by Kussmaul's experiments, but that this is the case in the real epileptic seizure is by no means proved. On the contrary, there are many reasons opposed to it (cf. loc. cit.). My opinion rather is that *the excitation of the vaso-motor centre and that of the centres for the muscles are co-ordinate;* that both go on side by side, and are independent of each other.

Upon this view alone do a series of otherwise incomprehensible peculiarities, such as are observed in many seizures, become capable of explanation. Among these may be mentioned, in the first place, the occurrence of epilepsia mitior (without convulsions); secondly, the occurrence of twitchings before the coma; then again, the fact that convulsions may occur wholly without loss of consciousness, although this very rarely happens; finally, the commencement of certain cases with cyanosis, caused by primary spasm of the muscles of the throat (trachelismus).

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[1] Whether the vaso-motor nerve discovered by *Benedict* in the floor of the fourth ventricle deserves consideration in connection with the circulation within the skull, and particularly in the epileptic attack, cannot as yet be decided in view of the novelty of the facts communicated by B.

What then is the stimulus which throws into activity the sets of apparatus which have been mentioned? In the cases where (*e. g.* in secondary epilepsy) pressure upon a cicatrix determines a seizure, it is incontestably the sensory excitement advancing centripetally; and quite in analogy with it a peripheral action upon a sensory nerve can often be made out; at other times it appears to be a mental impression, when the process of excitement advances downward from the cortex to the pons and medulla oblongata. For many cases, however, no determining influence of any kind whatever can be proved; the attack breaks out as the spark darts from a strongly charged Leyden jar, to use the simile of Schroeder van der Kolk. Hypotheses can be set up to account for these cases also, but we consider it judicious to suppress them, as they are only hypotheses.

In giving the history of the symptomatology of the seizure we give prominence to the necessity of distinguishing several periods in it. Now it is very probable that such a distinction obtains, not merely in the external sketch of the symptoms, but also for their pathogenesis. If, namely, we consider a patient in the so-called *second stage of the attack*, with the veins of the head and neck filled to distention, the face markedly cyanosed, and the eyeballs projecting, nothing supports the assumption of a cerebral anæmia, but everything points much more to an intense venous hyperæmia within the skull, which is the result of the spasmodic contraction of the muscles of the neck. The excess of carbonic acid in the blood which is present in the skull is then still further increased by the marked interference with the respiration, resulting from the spasm of the respiratory muscles. Further than this, we know from direct experiments (compare above) that great venous hyperæmia of the brain *may* produce general convulsions, and we also know the same from the symptoms attending the final act of life in suffocation.

From this the conclusion seems to be warranted that in the further course of the attack loss of consciousness and spasms are caused and maintained by venous hyperæmia. The possible objection that interference with respiration and venous stasis are not intense enough in the epileptic seizure to be capable of producing these serious results, appears to be without significance in view of the special conditions here present. For, as we have seen above, we are forced to maintain in epilepsy the existence of an abnormally increased excitability of the central nervous apparatus involved, so that even slighter irritations than usual are capable of setting it in action; and then it would also be possible that these systems of apparatus, if already thrown into activity, might be maintained in that state for a certain time by even a weaker irritant, although this cannot be actually proved.

This might be the proper place to discuss the questions of the way in which anæmia produces coma, hyperæmia convulsions, etc. We have already touched upon these matters in part in another portion of this work, *apropos* of cerebral anæmia and hyperæmia, and in part they are exclusively of a physiological nature, so that we must decline a further discussion of them here.

If the pathogenesis of the ordinary seizures can be very little cleared up at the present time in any respect proportioned to its prominence, it is very much more difficult to gain an insight into the processes which go on in connection with the abnormal forms of seizure, and with the epileptoid states. The idea which naturally suggests itself is to assume that in them also disturbances of the circulation in the cranium constitute the essential feature, and the same in the case of epileptic delirium and many epileptoid forms. An attempt at an explanation of this kind has been actually made by certain investigators, and we must admit that, according to our opinion, a solution of the riddle under consideration is perhaps to be looked for earliest in this direction, but we are moving meantime purely in the realm of speculation. In many cases, in those, *e. g.*, where patients in the seizure walk around in a state of unconsciousness, there is absolutely no point to be seized, even for an attempt at an explanation.

Closely related to the pathogenesis of the seizure is the question of the significance of the prodroma. *How is the so-called aura to be interpreted—how does it originate—in what relation* *does it stand to the seizure itself?* Opinions are much divided regarding these points, for, while some authors put the so-called "warnings" altogether aside, others attribute great importance to them. How far the latter view is correct in reference to treatment and diagnosis has been already or will yet be considered. At the moment the essential question for us is in regard to the relation of the prodroma, and more particularly of the "immediate" prodroma, to the seizure itself. Our own opinion in regard to this is as follows:

The immediate "prodroma" belong to the seizure as a part of itself; they are therefore rather symptoms, and not properly prodroma. In connection with this, too, it has already been said that they cannot, as a centripetal irritation, cause the outbreak of the seizure. We are much more inclined to believe that we must assume the origin of every aura to be central.

Unfortunately, in this place we cannot enter into a detailed proof of our view; we must limit ourselves to the presentation of certain leading points.

The possibility of this view must be conceded if, as we do not doubt, our idea of the origin of the seizure itself be correct, *i. e.*, if the excitation of the different "centres," located in the pons and medulla oblongata, be co-ordinate. Just as the excitation of the vaso-motor centre frequently precedes that of the "convulsion centre," and just as sometimes trachelismus sets in first (compare above), in the same way is it conceivable and possible that the excitation in the groups of ganglion-cells, which leads to the (spasmodic) innervation of an arm, a leg, or single muscles, may also occur as the first link in the chain of symptoms. The same thing is repeated in the circumscribed vaso-motor prodroma, among which we reckon the ambulatory seizures with vertigo and confusion which are so frequent, for these obviously represent only a lower degree, a first step, in the subsequent loss of consciousness. The sensory symptoms, too, may likewise be conceived of as eccentric, *i. e.*, caused by central processes; this view only requires to undergo a modification for the cases where a peripheral lesion has furnished the starting-point for the trouble.

In many forms of epilepsy the so-called prodroma, especially the motor ones, are already regarded as belonging to the seizure. These are particularly the cases in which a lesion of the cortex has caused the disease, and in which spasmodic phenomena always usher in the seizure in the same manner. Here one might be disposed to abandon this view as altogether impossible.

The attempt has been made to find a leading objection to the central origin of the aura in the fact that one sometimes succeeds by a so-called interruption of it, such as by putting a ligature on the extremity, etc., in preventing a further outbreak of the attack itself. This objection, however, is without any force, since, as we mentioned above, we are here dealing not with the interruption of a process of irritation which is creeping towards the centre, but with a "reflex inhibition." One can scarcely think of a more striking proof of this than Odier's case furnishes, which is cited almost everywhere (compare Romberg, and Portal). In this case the attack was ushered in by twitchings in limited groups of muscles; it could be repressed by compression of the arm—and post mortem a lesion of the cortex was found as the starting-point which caused the epilepsy.

On the other hand, no proof whatever has as yet been brought forward to show that, as Reynolds, *e. g.*, expresses himself, the aura should be regarded as a peculiar *peripheral* condition analogous to that already existing in the medulla oblongata, and it rather seems that this view is more difficult to reconcile with the genesis of the seizure and with the symptoms.

According to their prominence, the partial twitchings and the other phenomena, which so often occur in epileptics during the intervals without being followed by an attack every time, must be considered as incomplete seizures, after the manner of epileptic vertigo and the *petit mal*.

Finally, we must add a few words on *epilepsia vasomotoria*, which has of late been described as a special form. In general, we understand by this term those cases in which the symptoms of arterial vascular spasm are more strongly marked than usual, especially where they are observable for a longer or shorter time before the outbreak of convulsions and the loss of consciousness. From this, however, we cannot deduce the propriety of setting up a special vaso-motor form. For, in the course of our exposition, we have often enough insisted that only very few seizures run their course absolutely without vascular spasm; loss of consciousness is, in fact, always dependent upon it. It does not then at all change the nature of the thing whether a greater or a less peripheral extension of the vascular spasm occur before the appearance of the severe symptoms of the attack. So, if we would attribute special importance to this, we must likewise, if the coma has been preceded by rather decided partial twitchings, affirm the existence of an epilepsia motoria, or, in case of a marked sensory aura so-called, an epilepsia sensitiva, which certainly is not correct, or at all events useful.

At the same time, these cases with marked vascular spasm have their significance in reference to the theory of the seizure, inasmuch as they give essential support to the assumption of vaso-motor cerebral anæmia. The conditions described by Landois[1] and myself[2] as angina pectoris vasomotoria are particularly instructive in this connection. In one of my patients who was suffering from this affection, which is undoubtedly of vasomotor origin, there were developed, in connection with a decided sense of dizziness, slight clonic twitchings in the extremities; in a female patient who quite recently came under my observation there occurred almost complete loss of consciousness; and Berger[3] even noticed that in some instances the symptoms of the angina pectoris vasomotoria developed into a complete epileptiform seizure.

Course and Sequelæ.

True epilepsy is an eminently chronic disease; it lasts for years, in very many cases even to the end of life. It is a very exceptional occurrence when a genuine epilepsy disappears again after a few months. The conditions which were formerly described as acute epilepsy are for the most part something entirely different, and only present a certain resemblance to epileptiform seizures. It indeed sometimes happens that, by an unlucky accident, death occurs during the paroxysm, after the disease has existed for but a short time; but occurrences of this sort furnish no reason for denying the essentially chronic course of the malady. Besides, we have already declared that death in the seizure itself, and in direct consequence of it, is one of the greatest rarities.

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[1] Correspondenzblatt f. Psychiatrie. 1866.

[2] Deutsches Arch. f. klin. Med. Vol. III.

[3] Die Lähmung des N. thorac. longus. Habilitationsschr. Breslau. 1873. Note to p. 22.

When speaking of the frequency of the seizures, we treated of the causes which had an effect in giving rise to them, and in characterizing the course of the disease mentioned many points of importance, so that only a few details remain to be referred to. We are principally concerned with the question *whether there are definite influences which have a modifying effect upon the course of the disease.* Particulars bearing upon this have been communicated by various observers; Delasiauve, especially, has taken upon himself a careful discussion of the influences which here come under consideration. From the statements before us, with which our own experience agrees, it appears that in general the most different conditions, whether seated in the patient himself, or in the circumstances surrounding him, or in accidental changes beginning from without, or brought about in the organism itself, exert only a very slight modifying influence upon the course of epilepsy. And if certain conditions do act in this way, the effect is not uniform; *i. e.*, with the same condition, it may at one time be unfavorable and at another favorable. A more constant and essential influence can be proved for but very few causes. The principal of them are the following:

Excess in alcoholics almost always leads to an exacerbation or to the outbreak of attacks, even if spontaneously, or in consequence of medication, they have been for a considerable time held in abeyance; of course, this is not invariably so. It is also maintained by certain observers that even the moderate drinking of wine and beer is unfavorable. Relying upon these statements, I have always been accustomed to prohibit the use of alcoholics altogether, and in consequence I cannot judge of their influence from my own experience. The same obtains with regard to tea and coffee as with regard to alcoholics.

Excesses in eating also act not infrequently like those in the use of wine. Whether certain definite articles of food are more hurtful than others is questionable, or should be considered as depending on individual idiosyncrasies. It is certain that coitus sometimes induces seizures; it does not always do so, however, and, on the other hand, examples are known where the practice of it made the paroxysms temporarily less frequent. With regard to menstruation, we have already briefly expressed our opinion above; its effect is extremely variable. Sometimes, when the disease already exists, an aggravation of the seizures in frequency and intensity takes place with the setting-in of menstruation; sometimes they occur with special severity immediately before or after the period; again at other times the attack occurs only in exact coincidence with the menses. On the other hand, however, instances are known where an improvement in the disease, chiefly in respect of a diminution in the individual paroxysms, was established with the first appearance of the menstruation, or at the time of the separate periods, or where the disease showed improvement when menstruation, which had previously been irregular and painful, became regular, or even in certain cases entirely disappeared. Equally variable is the influence of pregnancy. We only add the fact, said to be sometimes observed, that women during pregnancy remain free from seizures when carrying a boy, but not when carrying a girl; or also the opposite.—Further than this, the influence of strong mental impressions upon the causation of seizures is indubitable; yet the real course of the malady is but seldom changed by them.—The question would be worthy of comprehensive statistical investigation, how other intercurrent diseases of various kinds influence the course of epilepsy. Up to the present a few general propositions hold in regard to this, and are comprised in the following: acute diseases, during their existence, generally result in a cessation of the seizures; only very exceptionally do they lead to a complete disappearance of the malady; at times this is witnessed even after accidental external wounds, burns, and the like. Chronic affections act in very different ways; at times they intensify, at times mitigate the seizures; at others, too, they remain wholly appearance, may interfere in every way with the social life of the patient; not merely from the serious mental disturbances which appear with them and in consequence of them; but also particularly from its rebellious obstinacy to treatment. The question has been propounded in all seriousness by some persons, whether epilepsy is at all curable. It is curable; that will certainly be quite generally conceded at the present day. With regard to the proportionate number of cures, views certainly differ somewhat. Most observers, however, are also agreed in this, that the percentage of cases cured is quite a small one. In general, therefore, the prognosis must be regarded as rather unfavorable. — We purposely refrain, in the following paragraphs, from giving figures, because no great value can be attached to them, as the series of observations of the individual authors were made from wholly different points of view; sometimes the cases were collected in institutions, sometimes in private practice; sometimes the conception of epilepsy is more strict, sometimes more liberal, etc.

In the first place, there are very isolated cases of spontaneous cure, in which the malady disappears without any treatment; they are reckoned at from four to five in the hundred.

In the second place, an improvement may be wrought by treatment to such an extent that the individual seizures come less often. This result is not so very unusual. A temporary improvement, indeed, will be quite frequently obtained, but it may also be permanent.

In the third place, some cases are actually completely cured. Certainly, very few observers have been able to verify the experience of Herpin, according to which some fifty per cent. of all epileptics would be curable. In general, complete cures may always be regarded as exceptions; but it cannot be denied that Herpin had actually shown a series of permanent cures, even if we were inclined to regard his own statement as too favorable, for, according to Voisin, the permanent cure of many of Herpin's patients had lasted for ten years after his death. To him undoubtedly belongs the credit of having again vigorously emphasized the possibility of curing epilepsy, and of counteracting the mischievous do-nothing policy.

Finally, into a fourth group, and that unfortunately the most numerous, fall all the cases in which every therapeutic endeavor remains without effect.

The following circumstances have an effect upon the prognosis in special cases, either for better or for worse:

The age of the patient at the beginning of the trouble; before the twentieth year the prognosis is, *cæteris paribus*, more favorable. Herpin regards those cases as having still better prospects which first begin after the fiftieth year.—There is also scarcely any difference of opinion on the point that the curability diminishes in the same degree with the duration of the disease. Herpin formulates this more fully still when he says that the duration in itself does not make the prognosis worse, but the number of seizures suffered in a given time; *i. e.*, the possibility of cure is greater when an epileptic subject has experienced twelve attacks in the year than when there have been a hundred and twenty. If there have been more than five hundred seizures, the prospect for recovery is equal to nothing. Opinions are at variance with regard to the significance of very long intervals; we regard them rather as favorable. Whether the attacks happen by day or by night can scarcely be of importance. Likewise we do not believe that the character of the separate seizures, whether epilepsia gravior or mitior, materially alters the prognosis with reference to curability.—In view of the variety of the particular etiological influences, it requires no long demonstration to show that they are of great and very different value in prognosis. Those cases have always been regarded as the more favorable in which a peripheral cause produced the epilepsy, for here, of course, the curability of this itself comes to be a question of the first moment. But even if the condition just named exists, we must always be quite circumspect if the epilepsy has already lasted for a long time, and we cannot count implicitly upon restoration to health. Central causes render the prognosis very unfavorable. Still, if we do not have to deal with material lesions, but with a mental influence merely, the affair assumes a somewhat better shape. Hereditary epilepsy is generally esteemed incurable; yet Herpin and Reynolds, *e. g.*, have seen marked exceptions to this rule. The same holds, according to the latter observer, for the disease when due to masturbation. Reynolds lays it down as a general proposition that those cases are least amenable to treatment in which the source of the disease is involved in the greatest obscurity.

The question of what significance attaches to the frequency, the form, and the combination of the seizures, and various other influences having reference to the development of mental lesions, has been already discussed above.

Treatment.

Small, indeed, is the actual encouragement derived from looking through those chapters of medical literature, from the oldest to the most recent times, which refer to the treatment of epilepsy. The methods and the remedies change; but the final result always remains the same beggarly one. There is no single article which acts with anything like an approach to the certainty of, *e. g.*, quinine in malarial diseases, no single method of treatment which could be even approximately compared with, *e. g.*, the electrical treatment of traumatic peripheral paralyses. Many remedies and methods of treatment have isolated successes to show, but nothing is to be depended on; nothing, even on careful discrimination of cases, affords a sure prospect of recovery, or even of improvement. From time to time it has been supposed that a panacea has been found, or a veritable curative process discovered, until the reaction comes again all too soon. Then we are the richer by a remedy which in favorable cases shows a few results in recovery, or improvement, more than its predecessors; but the search begins anew.

By this we do not wish to say that we regard better hopes for the future as illusory. This is just as little allowable as it would have been before the discovery of the itch insect and the introduction of the balsam of Peru to be willing to deny the possibility of the absolutely certain process for curing scabies which now really exists. For the present we must unfortunately content ourselves with the existing remedial agents. But the more insufficient these are, and the more authoritatively they are recommended, so much the greater is our duty not to manage all patients on one model, but here, as everywhere in the field of therapeutics, to proceed on the one correct *principle of individualizing*. The physician who treats every epileptic who comes under his observation indiscriminately with whatever happens to be the prevailing fashionable remedy, with nitrate of silver or bromide of potassium, with oxide of zinc or valerian, with gymnastics or hydropathy, will have fewer successful results than are generally attainable.

The therapeutic measures in each individual case have generally a threefold object. They resolve themselves into a causal treatment, a general and dietetic treatment directed to the nervous system, and the employment of definite medicaments.

The *causal treatment* is unfortunately possible in very few cases, as appears at once from a review of the etiological relations, which are generally so obscure; but where a causal influence accessible to treatment can be found, efforts must be made to remove it. At the same time experience teaches us that, even in case of the removal of the assumed or actual source of the disease, this itself does not always disappear. The morbid state in the pons and medulla oblongata is already so far advanced that it presents itself as an affection wholly independent of the primary source. A knowledge of this fact is important for the avoidance of prognostic errors and therapeutic illusions.

Primarily the causal treatment claims consideration in those cases where a peripheral lesion shows itself as a starting-point. Literature recognizes a great number of instances where, by the extirpation of a cicatrix, of a tumor pressing upon a nerve, or by the opening of an abscess, the epilepsy was caused to disappear. The same is true of a series of pathological conditions in the internal organs. All the varieties which are possible here cannot, of course, be cited in detail; a careful review of the history, a thorough examination of the patient, must lead to the right course in each individual case. We would only call special attention to one point: formerly, in case an aura was very pronounced and constantly recurred in the same fashion in the course of the same nerve, not only was the suspected finger, but even the hand or arm amputated, or neurotomy performed, without thereby curing the epilepsy. To-day it would be only in the most exceptional cases that a particularly bold operator would venture on so violent a step as this, especially as it is known that the most typical forms of aura may be of central origin. It could be thought of then only when there was great probability that a peripheral wound furnished the starting-point for the disease. But even in these cases we would caution against the severe operations with removal of extremities, because it can never be known whether even after them the epileptic change will not remain behind.

It may be observed, by the way, that those cases where the disease persists after neurotomies and amputations seem to us very much to favor the opinion advanced above of the eccentric nature of the aura.

The practice of *trephining* is also to be reckoned with the causal treatment. Although adopted in former times most extensively in every case which resisted medical treatment, and although warmly recommended by Tissot, in the course of time it has been continually less and less frequently practised. It is certainly true that in individual cases the disease was cured, and these brilliant successes, rare though they were, have always been an incentive to further trephining ; even Echeverria reports three striking cases, regarding the ultimate effect of which, to be sure, nothing can yet be said, because an immediate arrest of the seizures proves nothing. But even if it must be conceded that all the favorable cases were not those merely of symptomatic epilepsy, that, in other words, trephining *may* become a causal remedy (*e. g.*, if exostoses from an injury to the cranial bones have led to true epilepsy), though not necessarily so, any more than in the case of excision of a peripheral cicatrix, still, in any given case, all influences should always be most carefully weighed, as an absolutely certain diagnosis can never be established even under the conditions that we are here considering. Consequently, we cannot with Hasse "urge that any indication whatever that warrants trephining should be followed," but we are willing to grant that the operation, in accordance with our present experience, is generally allowable, and we must regard its success in any particular case as a piece of good fortune, which could not have been predicted.

The causal treatment must take another direction if general constitutional anomalies have caused the epilepsy or led to its outbreak. The measures to be taken against drunkenness, rachitis or scrofula need not here be particularly detailed. When general plethora or anæmia is present the question of its treatment is more important. In respect to both of these, individual observers have overshot the mark, though in the former more than in the latter. At the present day we are agreed as to the essentials in this matter, viz., that with epileptics anæmia and especially a weakly constitution always require an appropriate treatment, the details of which are well known; that plethora, on the contrary, requires direct medical treatment only when present in a very marked degree. In the latter case the familiar curative procedures appropriate to the case should certainly not be neglected, and among them in certain cases the abstraction of blood may even find a place; yet the need of a lowering mode of treatment, even in the beginning of the disease, must always be reckoned among the exceptions. It is less to be feared that any one will fall into the opposite error, committed by Radcliffe, of using a tonic treatment for every epileptic indiscriminately.

The causal treatment directed to other etiological relations is still more impotent than in the case of those already mentioned. Where a decided mental change has led to the disease or brought about an outbreak, the attempt has been made to effect a curative action by a similar one, as by sudden fright, by threats, and the like. But who is in a position to estimate the results of these? And in opposition to the few cases, on which the authority for such a procedure mainly rests, where an accidental violent mental emotion produced an improvement or arrest of the epilepsy, there stand the much more numerous instances where they have caused a decided aggravation. For this reason also the best physicians have constantly advised against them.—Finally, the removal of an inherited tendency may be the only object of prophylaxis. Romberg thus expresses himself in reference to this: "in families where epilepsy is a pathological entail, marriage of the members among themselves is to be prevented, and the veterinary principle of crossing with thorough-bred stock is to be introduced." According to what has been said already, this advice must be extended not only to epileptic persons, but to the neuropathic in general.

Apart from those enumerated, there may still occasionally be various separate influences which demand therapeutical consideration, the detection of which must be left to the skill of the physician, as general rules cannot be laid down for them. Thus, *e. g.*, it has been determined that epileptic seizures occurred with greater violence and frequency when a cutaneous eruption of some standing has been healed, and that they disappeared on the re-appearance of the exanthem. These and analogous relations must always be carefully considered.

If we are not successful in getting at any causal indication, the treatment of the central epileptic change must be attempted. To this end we make use of dietetic measures, and, in addition, of curative procedures, which are either to act locally upon the brain in the pons or medulla oblongata, or to influence the nervous system at large, and, finally, of medicines to which are ascribed either an effect in subduing "the exalted irritability" of the central organs, or, more vaguely, a "specific" relation to epilepsy.

For *the regulation of the diet*, in a stricter sense, the eating and drinking, very positive indications are sometimes presented to us, as in pronounced anæmia or plethora. Thus we have already expressed ourselves to the effect that alcoholics, tea, and coffee must be entirely forbidden to epileptics, or—in case this is for any reason inadmissible—should be allowed only in the very smallest quantities. The disadvantages, too, of luxurious and sensual living have already been insisted on; in general terms patients are always better off for a temperate and well-regulated mode of life. Cheyne relates the instance of an epileptic physician, in whom the seizures occurred so much the more rarely, the fewer and more easily digested were the things he ate, and who, in consequence of this observation upon himself, limited his daily nourishment to water and about three and a half pints of cow's milk; this he continued, as stated, for fourteen years and recovered. An exclusively milk diet could, however, be long continued, only in very exceptional cases. Tissot gives a selection of the various aliments which are suited to or injurious to epileptics. Still, in this respect one is, in part, to be guided by the different individual peculiarities, and much must be made dependent upon the observations made by the patients upon themselves. As a general rule, it can only be laid down that if no direct indication for an invigorating procedure is offered by the condition of the patient, a preponderance of vegetable and milk diet is to be observed.

Light, not exhausting mental effort, and the avoidance, as far as possible, of emotional feelings, are essential adjuvants in treatment, according to all that has been said above. It is quite wrong, however, to attempt to forbid mental work entirely to epileptics; on the contrary, a moderate, quiet exercise of the mental faculties is, as a rule, very advantageous. The same is true of physical labor; walks and simple gymnastic exercises, free from danger, are very good for patients, only they must not be carried to complete fatigue. We even know of certain instances where epileptics have recovered by a mere change in their mode of life, where, *e. g.*, instead of a sedentary in-door life they have taken to gardening.

I must, from my experience, regard *water-cures* as a very important means in the treatment of epileptics. Through them I have seen, although not complete recovery, at least a very essential improvement take place. To be sure, I cannot claim this for all cases, but those in which no effect at all was visible were very old, inveterate epileptics, in which frequently a failure of the mental powers existed at the same time. Still, in these hydro-therapeutic doings one must not be satisfied with merely being rubbed down at home, but the treatment must be methodically carried out for from six to twelve weeks in a cold-water cure institute. It is most judicious to leave the kind of hydro-therapeutic treatment to the physician of the establishment, and I would only insist in general, that I regard douches upon the back and head, and plunge baths, as rather injurious than useful. With reference to the selection of the institution it depends less upon the geographical position than upon the physician who conducts the establishment. It is readily understood that discretion, no less than the changes taking place in the medical personnel of the establishment, prevent our giving more positive statements in regard to this. Only a marked degree of anæmia should be looked upon as a contraindication to the water-cure.

River and sea baths are far below the methodical water-cure in efficiency; besides, there should be considered against them a circumstance which, although extrinsic, is still always worthy of attention, to wit, the danger of drowning, to which patients are exposed if they are overtaken by a seizure while bathing without special supervision.—As to *warm baths*, I have no experience at my command; yet, according to the existing statements of other authors, no special benefit is to be expected from them.—Here, too, we must mention the *treatment recommended by Chapman*, by means of the application of ice-bags along the spine, the details of which are well known. Beigel declares in general terms that Reynolds has seen "excellent service" from them; Chapman describes several instances of decided improvement. In several cases where I have tried this method in epilepsy, I can boast of no noteworthy result.

A great deal was expected from *electricity*. New experiments were made with every new improvement in the apparatus; the curative effects, however, in epilepsy always remain insignificant, and therefore we believe we may omit any account of the separate experiments. When the constant current was first introduced in the shape of improved apparatus (Remak), its use also in epilepsy was recommended from the most different quarters. All thoughtful electro-therapeutists are agreed, however, that even the constant galvanic current accomplishes only very moderate results; and meantime it should not be lost sight of that in many instances other curative agents were employed in connection with electricity, or at the same time with it. I myself, even after long-continued use of it, have never observed anything more than an improvement as the most favorable result, but no perfect cure. Still, we cannot doubt, in view of the definite statements of certain authors, that sometimes a cure can be effected by the current, so that, of course, we must not push our scepticism too far, although it derives some support from the fact that the constant current has not been in the hands of physicians much longer than from ten to twelve years. At all events, however, it is certain that we should not expect too much from electricity.

The application of the current is usually made in numerous combined ways. At first the electrodes are placed high up on either side of the nape of the neck, so as to act upon the medulla oblongata and the pons; afterwards galvanization of the cervical sympathetic is generally practised. According to the particular case, we may combine the transmission of currents transversely through the cranium, or through the spinal column, or the treatment of special nerve-tracts, in which an aura has been particularly pronounced.

In former times, especially, great weight was laid upon the establishment of so-called *counter-irritations*, notably in the nape of the neck. This was done in the most various ways, from dry-cupping to the seton. The most famous advocate of the seton in recent times has been Schroeder van der Kolk. Meanwhile, the majority of observers have tried this and other counter-irritants quite in vain; we have ourselves introduced setons in some cases without observing the slightest change in the disease. Some advantage might be expected from the slighter counter-irritants, but only where there was considerable hyperæmia of the brain during the intervals.

In passing, two curative procedures may here be mentioned, which, to be sure, are no longer used, but each of which has in its time played a great part; these are ligation of the carotid and tracheotomy. We know to-day that neither of them can cure epilepsy, and they have been abandoned as dangerous operative procedures. It may be allowable to give a single historical notice of tracheotomy, for which, indeed, the recommendation of M. Hall is brought forward. This writer himself, however, in his earlier works[1] mentioned tracheotomy as a remedy only "against the most extreme degree of coma after the epileptic seizure," and then says, "but it is a strange idea that tracheotomy could prevent the epileptic convulsion."

We now come to the section on so-called *specific remedies*. Every one who writes on epilepsy feels himself tempted in this chapter to renew the old complaint of the glaring contrast between the immense number of remedies recommended and the extremely few cures effected. Still, we would leave general considerations wholly aside; they can lead to nothing here. Only the question may be raised whether it is essential to the interest of the subject, and of our exposition of it, to adduce here by name the hundreds of medicinal substances which have ever been tried for the relief of epilepsy. We do not at all believe so; whoever is interested in this may go over the separate names in the different monographs, among which we mention particularly those of Loebenstein-Loebel, Portal, Delasiauve, and, besides these, the chapters treating of epilepsy in Tissot and Joseph Frank. It appears to us really more proper as well as more in the interest of the practical physician, to mention only those articles as to which we have a more general experience, those in which the best observers recognize a considerable efficiency, and of which they have had more than an ephemeral experience. One may also without harm disregard a special grouping of the articles.

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[1] *E. g.*, On the Diseases of the Nervous System. Translated by Wallach. Leipzig, 1842. pp. 390–1.

First, as one of the oldest remedies, we mention the *radix valerianæ.* The most famous observers, especially those of the last century, have insisted on its usefulness, and it would be considered as indulging in unwarranted scepticism if one were unwilling to concede, in the face of the very positive statements which are made, that essential improvements, *i. e.*, absence of the seizures for years, and even individual recoveries, are achieved with the valerian root. We can ourselves give no decisive verdict on this matter, for the reason that we have not employed it alone, but always in combination with other substances.—Upon what its curative properties in epilepsy depend, no satisfactory explanation can be given. At the most, the experiments with oil of valerian, made very recently by Grisar,[1] might furnish an indication, for the frogs, to which 0.02 gramme (three-tenths of a grain) of it was given, became quiet and apathetic, exhibited a diminished reflex-irritability, and finally became completely stupefied, but gradually recovered. The diminished reflex-irritability appears to depend upon an influence affecting the spinal cord as well as the centres in the brain which excite spasm.—Practically it would certainly be of the highest importance to know the special conditions, so as to be able to determine accurately the cases in which valerian would be of more service than other articles. Unfortunately, this too is wholly unknown; for the older statements in regard to this are much too indefinite, and there are none more recent. As to the different preparations, the root has been given in the form of powder, or as infusions, extracts, and tinctures. According to my own experience, which agrees with that of others, the form of powder is the most appropriate. The doses at first should be small, about 0.5 gramme (seven and a half grains) three times a day, and should then gradually be increased to a total daily quantity of 25 grammes (about six drachms).

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[1] *V. V. Grisar*, Exper. Beiträge z. Pharmacodynamik der ätherischen Oele. Bonn, 1873. p. 62.

In the same category with valerian we place the *radix artemisiæ* (vulgaris). The wormwood was given in ancient times, and again in the first decennia of this century. It too is very apt to prove inefficient. Still my experience with it in some cases has determined me to bring forward the remedy here. These were cases where almost everything had been tried without the least benefit. When I finally had them take the radix artemisiæ, after a short time a decided remission in the number of the seizures became manifest. I lost sight of the patients, so that I can make no report of the final issue, but even the result stated seems to me an improvement worthy of note, especially after zinc, silver, atropine, bromide of potassium, valerian, electricity, etc., had been given entirely without effect. Besides, my observations were on patients at the time of puberty, in whom epilepsy had developed without hereditary predisposition or other ascertainable influences; in a girl of about sixteen there existed marked molimina menstrualia. The old recommendations praise wormwood especially in the epilepsy of women with demonstrable lesions of the genital apparatus. I direct the remedy to be used in the form of an infusion, 15 grammes (three and three-quarter drachms) to be taken daily.

No other vegetable remedies are the least worthy of mention; the improvement or cures obtained through them are so isolated that it is quite impossible to judge whether we have to do with accidental occurrences or with a causal connection. Only a few substances belonging among the narcotics furnish an exception to this.

Belladonna was praised even by the earlier physicians (Stoll, Theden, Hufeland, and others), and there are numerous cures described which are stated to have been attained by it. One of its most zealous advocates is Trousseau. Recently, instead of the herb, the alkaloid, *atropine*, has come into favor. It is said that recovery has been brought about by this too. Although we ought not to disregard the positive statements of certain authors, still we cannot from our own experience join in the praise of this article except to a limited extent, inasmuch as we have, to be sure, sometimes been able to effect an arrest of the seizures for a month by injections of atropine, but never a cure. The experience of others agrees with this, *e. g.*, that of Reynolds, who obtained by means of *belladonna*, which I have never given alone, an amelioration in the case of various patients (a cessation of many of the indefinite troubles of various kinds from which epileptics often suffer, such as nervous uneasiness, trembling, and disturbed sleep), but no complete recovery. The valerianate of atropine, from which certain persons had great expectations, seems to possess no advantage over the other salts of the alkaloid. Just the same is true of *hyoscyamus* as of belladonna.

The remaining narcotic substances, almost all of which have been tried at times, are actually of no more service, in fact are less serviceable than deadly nightshade and henbane, and may therefore very well be entirely dispensed with. In regard to *opium* in particular, it is generally recognized that it is of no benefit in the cure of epilepsy, but it may be most opportunely employed according to general indications in combating certain manifestations symptomatically. Hasse, as well as Morgagni, witnessed benefits from it in nocturnal seizures, if the patients were not plethoric and were freely purged beforehand.—*Inhalations of chloroform*, too, may be added here, with regard to which it is certain that they accomplish no curative effect. A difference of opinion even prevails as to their utility in respect to the occurrence of the individual paroxysms, since, on the one hand, they have been seen to delay the development of the paroxysms and limit their intensity, and, on the other hand, to act in exactly the opposite way. From which it may be safely inferred that chloroform inhalations are not of essential benefit.

In the list of metallic remedies, the most various have likewise been tried. The majority are acknowledged to be wholly inefficacious. To only a few do different authors ascribe a certain action in epilepsy, and in regard to one alone there prevails a greater degree of unanimity regarding its curative powers, although there is wide divergence of opinion as to their degree. This one remedy is the *oxide of zinc* (flores zinci). Having been first ordered now and then in the last century, it acquired a great reputation, especially through Herpin, although Hufeland also esteemed it very highly. Herpin relates that he cured twenty-eight out of forty-two individuals by the oxide of zinc. This result, which Hasse rightly remarks upon as "unprecedentedly" favorable, necessarily prompted to the decided employment of the remedy. Unfortunately, later observers could not corroborate this; but, as we have already had occasion to remark above, it would be quite incorrect for us on this account to dispute altogether the results obtained by Herpin, for recently Voisin has established by investigations the lasting character of the cure in several of the patients treated by Herpin; and, in fact, the different observers always assign a favorable action to zinc above any other of the older remedies. My own observations are not uncomplicated, because I have always ordered it together with other therapeutic measures; but if it be allowed in a limited sense to draw conclusions from such mixed methods of treatment, I could plead decidedly for the retention of the oxide of zinc in the materia medica of epilepsy. I have also, with Herpin, found the remedy more efficient with patients under twenty years than in those of more mature age. I ordinarily begin with small doses, three centigrammes (half a grain) at a dose three times a day, and increase gradually up to from 1 to 1½ decigr. (a grain and a half to two and a quarter grains) at a dose. If no unfavorable symptoms on the part of the digestive tract are produced, we may occasionally give even somewhat larger doses. Only when its use has been continued for some four months without the least effect, do I discontinue its further administration; otherwise I have given it for from six to twelve months. Usually I order the oxide of zinc in the combination in which it is given in the old pulvis anti-epilepticus, *i. e.*, in connection with radix valerianæ and the extract of belladonna or hyoscyamus.

Of the other metallic substances, only the ammonio-sulphate of copper and the lunar caustic deserve mention. The *ammonio-sulphate of copper* has been much longer in use than the oxide of zinc, but is very seldom given at the present time. If recent observers deny it the efficiency of the latter, their testimony is, as I have already insisted in another place,[1] simply insufficient, in the face of the statements positively made by the older authors. The difficulty consists in determining on the particular cases for its employment. I have there (loc. cit.) compiled what can be gathered from the published communications in regard to this. "If ammonio-sulphate of copper has led to a remission of the seizures or to a cure, it has done so much more generally with adults, less often with children (the reverse of the action exerted by zinc). The cases seem to comprise generally torpid, phlegmatic individuals, and not so much 'the irritable and nervous.' A necessary condition for the administration of the remedy, which is not to be given on an empty stomach, is a good state of the digestive apparatus." Moreover, I will not neglect to add that I have myself given the preparation in only a few cases, and then without noteworthy effect, after various attempts at a cure had already been made.

Nitrate of silver for a long time played a great part in the treatment of epilepsy, and certain of the older observers believed that they had effected more cures by it than by any other remedy. This opinion has very much changed. Of course, as in regard to the ammonio-sulphate of copper, the positive statements of earlier writers cannot be simply denied. Of course, the lunar caustic is sometimes given even at the present day; but confidence in its curative power is very much shaken. In the cases under my own observation, just as with the ammonio-sulphate of copper, I have myself obtained no convincing result; on the contrary, I can subscribe word for word to the remark of Reynolds, that patients have come to me whose skins were colored of a blackish blue by the use of the article for years, whose epilepsy, however, remained unchanged. In my opinion, the lunar caustic may at times be ordered experimentally, when the better approved remedies and modes of treatment have produced no result.

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[1] My Handbuch der Arzneimittellehre. Berlin, 1874. 2d Ed. p. 335.

We now turn to that medicine which in the last ten years has undoubtedly been given to most epileptics; that is, *bromide of potassium*. Some praise it as the sovereign remedy against the disease; others say that they have observed scarcely more than transient benefit from it in isolated cases; the majority, however, place the bromide of potassium, at all events, higher than all anti-epileptics which we have hitherto possessed. The literature relating to it has already become so considerable that we think we can venture to make a summary of the general verdict, and in doing so must abstain from a citation of the different authors; our own experiments are comprised in what follows.

Bromide of potassium is not an infallible, sovereign anti-epileptic, but it is certainly of more service than all other remedies. A small number of cases are cured (so far as we can speak of curing in view of the as yet brief existence of this treatment[1]); another group resists any action of the remedy—the disease remains entirely unchanged; a third, and this the largest number, experiences a more or less marked improvement.

Although some authors do not acknowledge the cures, or, at any rate, have not themselves observed them, still all, with the exception of a very few, are agreed in this, that bromide of potassium makes the seizures less frequent, and, where they have previously been frequent, extends the intervals to several months, and even longer, without their then recurring in accumulated numbers or greater intensity. Even this result is of extraordinary value, as every one must allow, in view of the frequent failure of our other remedies and methods of treatment, and it assures to bromide of potassium a place in the therapeutics of epilepsy.

The arrest of the seizures sometimes coincides with the very beginning of the treatment, which is scarcely the case with any other remedy. It is true they sometimes set in again at once if the remedy is omitted; but the positive statement is made by various observers, and very recently also by A. Otto,[1] that frequently the mental disorders of epileptics undergo a decided improvement at the same time, so that patients may even return to a normal condition from a state of commencing idiocy.

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[1] With regard to the cures, I have spoken sceptically in my Pharmacology, 2d Ed.; yet it seems to me—retaining the limitation in the text above—that this is now scarcely open to question, in view of the numerous communications which have since appeared, and which have been made privately to me.

At the same time, as we would once more remark, it should not be forgotten that even bromide of potassium may at times remain completely inactive, and this I must corroborate from a series of instances that have occurred in my own practice.

In the beginning it was believed that in certain forms the bromide of potassium was more efficacious than in others. The more extended becomes the material for observation, however, the less proof is there of this. Etiology, duration of the disease (up to certain moderate limits), the frequency, form, or absolute number of the seizures which have already taken place, seem to exercise no influence upon the possible action of the bromide of potassium.

Questions as to the mode of action of the bromide of potassium, and what the real therapeutic agent in it is, whether bromine or the alkali, we may here be at liberty to leave unanswered; these belong in the special books on pharmacology. Special emphasis, on the other hand, should be laid on the mode of employment. Almost all observers are agreed as to these two points, that the remedy must be given for as long a time as possible continuously, and in large doses. In adults we begin with five grammes a day, and increase to ten or fifteen; when there is great tolerance of the preparation, even to twenty grammes (five drachms) a day. It is, of course, understood that there must be breaks in its administration if the well-known pathological accompaniments set in, such as disturbances of the digestion, diarrhœa, acne, and furuncles. When of great intensity, they may occasionally necessitate a complete withdrawal of the remedy.

In cases where the bromide of potassium alone has been of very little or no service, an effect has sometimes been gained by certain observers from combining it with another remedy; thus bromide of potassium has been combined with oxide of zinc, with conium (Echeverria[1]), and with other articles. Of these various combinations we ourselves have tried only that with Indian hemp, according to Clouston's method[2] of administering it in psychoses, but have been able to gain no positive conviction of its greater efficacy.

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[1] Archiv f. Psychiatrie u. Nervenkrankheiten. Vol. V.

With this we close the enumeration of anti-epileptic remedies, and will only just briefly summarize the manner in which *the curative measures should be conducted when a patient comes freshly under treatment.* First and foremost, after very thorough investigation of the etiological relations and an examination of the patient, comes if possible a causal treatment. If this is not possible, or if it does not attain the object, then a general dietetic treatment should be instituted (which, of course, must have already been partly brought into use during the causal treatment) according to the rules laid down above. Add to this the administration of bromide of potassium, with ultimately the employment at the same time of the constant current; then in summer a cold-water treatment in case there is no special contra-indication to its use. If with these measures, after perhaps the combination of the bromide of potassium with cannabis Indica or conium, no effect appears after a considerable time, the bromide of potassium and electricity are to be abandoned, but the general dietetic management is to be observed, and the cold-water treatment may even be repeated; at the same time we should now give oxide of zinc with valerian root and extract of henbane or deadly nightshade. If all these things have been tried in vain, any one of the other remedies mentioned above may now be tried, or some one of the immense number of those that are elsewhere recommended. In order to make things complete, we simply set down some of them here: assafœtida, castoreum, stramonium, aconite, digitalis, squills, animal oil of Dippel, oil of turpentine, quinia, phosphorus, strychnine, selinum palustre, gratiola, etc., etc.

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[1] Philadelphia Med. Times. Nov. and Dec., 1872.

[2] Referred to in the Centralbl. f. d. med. Wissensch. 1870 and 1871.

The Treatment of the Seizure itself.

It would, of course, be of considerable importance if we were able to ward off each individual seizure. Unfortunately, this is practicable only in rare cases. It is wholly impossible in cases where the paroxysms break out suddenly ; the full paroxysm may sometimes be prevented, but only in cases where some considerable time elapses prior to the loss of consciousness, and where the so-called premonitions precede it for a length of time. To accomplish this, a firm ligation of the extremities has at all times been in general use in those where a distinct aura showed itself. Possibly the following will also succeed—after the analogy of experiments on animals—if, in case the seizure, *e. g.*, is ushered in by an infolding of the fingers, we attempt to bring them at once by a rapid and forcible action into a position of hyperextension. (We need not lay any further stress on the fact that the senseless forcible extension of the thumb when a seizure has already occurred is not to be confounded with this.) There are quite numerous cases of this kind indicated in the literature of the subject.

Now, if such premonitory symptoms are absent, the suppression of the seizure will be much more difficult. Formerly, compression of the carotids was proposed with this object. Kussmaul has already exposed the untenable character of the theoretical arguments on which this was recommended, and Hasse has shown the practical difficulties in carrying it out, which are besides quite palpable. Favorable results from compression of the carotids have also been reported only with extreme infrequency. —The uncertainty of the inhalation of chloroform, to which that of ammonia is very analogous, we have already considered above.

Very recently the inhalation of *nitrite of amyl* has been recommended for cutting short the paroxysms. If we consider the usual mechanism of the seizures on the one hand, and on the other the physiological mode of action of the nitrite of amyl, as far as this is yet establishe mmendation is *à priori* perfectly correct, and the ef t have also been actually reported. At all e of nitrite of amyl at the very beginning of the seizure are worthy of further trial. We would only refer very decidedly to one point in this connection. This remedy should only be tried where the patients become pale at the very beginning, where indications of cerebral vascular spasm are present. It should not be administered if the color of the face is cyanotic from the outset. Further careful observation is required to teach us how it behaves when the facial color remains unchanged from the beginning.

"The seizure, when once begun, wears itself out; we may banish the idea of interrupting it, for the well-being of the epileptic patient is greater the more complete the seizure, particularly after a long interval has elapsed." This dictum of Romberg has still to-day its complete significance. The external measures, the object of which is to protect patients from injuries, the loosening of tight bands when possible, and the like, require no special description in this place. They are governed by the external circumstances in which the patient is overtaken by the attack. Unfortunately, it is but seldom possible to prevent the biting of the tongue. If the seizure has already broken out nothing can be thrust between the teeth. And even if the patient has time enough to put some kind of wooden wedge between his teeth, wounds of the tongue are not always avoided. In such cases we must always be careful lest objects of this kind get into the throat during the convulsions and produce suffocation.

As the comatose stage often lasts for some hours after the paroxysm, it is necessary in this, should the patients present no threatening symptoms, to guard against all excessive officiousness, or rather to do nothing, but leave the patient quietly to his sleep. Active interference is demanded only where the paroxysms follow each other in rapid succession, and where the status epilepticus is developed. Unfortunately this is often of no avail. Of the various remedies employed, blood-letting, either local or general, when there is strongly-marked hyperæmia of the brain, seems on occasion to be the most useful. Cold to the head, purgatives, and the most various internal medicaments are quite inefficacious. When the paroxysms are very violent, a careful trial might be made of inhalations of chloroform. The most approved remedies are to be employed against any threatening œdema of the lungs, which may develop, such as large blisters upon the chest, or acetate of lead in large doses given at short intervals.

Simulation of Epilepsy.

It is well known that epilepsy is the disease which is perhaps the most often simulated; military surgeons especially and those occupied with medico-legal cases have frequent opportunity of verifying this. The free intervals and the striking, frightful form of the paroxysms, which at the same time excite sympathy, are the chief influences which induce impostors to make this particular malady the object of their study. The endeavor has therefore always been made to find indications for the diagnosis of real from simulated attacks; very numerous instances, however, teach us that this distinction is not always easy.

We refrain from enumerating here all the points which have been assigned as capable of furnishing differential proof, because, with few exceptions, *none* of them have this character. It proves nothing that the imitator should choose time, place, and surroundings for the seizure, that he should fall down in the way he might be expected to, that he should turn his thumbs in again if they have been forced out, and do many other things, because they may also happen in real epilepsy. The physician will rather need to make use of a variety of attending circumstances, and must avail himself of much acuteness in order to get at the truth, and then it is sometimes amusing to see into what awkward traps persons fall who have produced seizures most true to nature. The experience of every physician versed in this matter will furnish to order a collection of anecdotes bearing on it. But if the impostor is cunning enough not to lay himself open, even if he has to endure painful procedures, which were formerly carried almost to cruelty, what then? Are there no symptoms of the seizure which *cannot* be voluntarily imitated? *Sometimes* there really are such, and any paroxysm may be regarded as genuine in which the following symptoms show themselves: pallor of the countenance and dilatation of the pupils at the beginning of the seizure, failure of the pupil to react when a bright light falls upon it. But, unfortunately, these trustworthy tests cannot always be turned to account, for the *opposite* conclusion, that every attack in which these signs are absent is simulated, is *not correct.* In the section on Symptomatology we have spoken in detail on these points.

It would be of the highest significance for the diagnosis of simulated epilepsy if the assertion of Huppert were confirmed, that transient albuminuria occurs after every pronounced attack. Still, we must first wait for the confirmation of this. Equally uncertain is the change in the sphygmographic trace of the pulse, on which Voisin lays stress for purposes of diagnosis, and which is said to occur only after genuine seizures (compare above).

ECLAMPSIA.

J. P. Frank says, in defining this disease, "eclampsia, epilepsia puerilis, a priori (sc. epilepsia) differt, quod acuta sit, incedens cum febre continua, interdum cum intermittente." Since then the conception of eclampsia has so far changed that Hasse used it simply as a synonym for "acute epilepsy," *i. e.*, those forms of spasm are denominated eclampsia which resemble epilepsy in their outward character, but which occur only a few times, passing rapidly away, and end either in death or in recovery.

Good observers, and among them Hasse himself, have now excluded from eclampsia a series of spasmodic attacks, although in external features they bear a great resemblance to those of epilepsy; in particular, all those convulsions which are simply the expression of gross anatomical changes in the nervous system, especially in the brain. Nevertheless, even at the present day, at least in German literature, the appellation "eclampsia" is quite generally used as a collective name to designate epileptiform spasms which appear under very different conditions, provided only that they be acute, soon disappear again, and do not subsequently return. In consequence of this the same chaos reigns to-day regarding eclampsia as formerly prevailed about epilepsy.

We are of opinion that we should go decidedly further, and designate by the ancient name of epileptiform seizures all the convulsions which appear simply as a symptom in the course of various other diseases, even if they be only acute and transitory; or, in order still less to commit ourselves in advance, that we should use the simple expression—general spasms. Just as the titles colic and icterus have disappeared from special pathology, and have been relegated to general pathology, so with advancing knowledge must we continually take away from that which was once, and still is, reckoned as eclampsia; very many of the processes once included under this conception have already been recognized as simple symptomatic convulsions, the process at the foundation of which can be very easily made out, and of course these must accordingly be treated of in the symptomatology of these fundamental diseases. Among them belong convulsions from hemorrhages or other rapid and profuse losses of nutritive fluids, especially in children, as also those which take place in cases of very great hyperæmia of the brain, in cases of cerebral or meningeal hemorrhage or embolism, or sometimes in the course of cerebral tumors and abscesses. The physiological connecting link, which is common to most of these cases, is cerebral anæmia; for the special data in regard to this the reader may compare the sections treating of it in the present work. It does not occur to any one at the present day to speak of eclampsia in such a case.—Another group, too, which formerly furnished a large contingent to the "cases of eclampsia," has been separated; we refer to the convulsions occurring in the course of diseases of the kidneys. It is not our purpose in this place to enter more minutely into the still vexed question of the way in which the convulsions and the cerebral symptoms generally arise in renal diseases, whether by œdema together with anæmia of the brain, or by the overloading of the blood with excrementitious matters; this likewise belongs in other places in this work. At any rate, however, it is superfluous and confusing to apply a special name, like eclampsia, to a manifestation which does not at all occur as an independent disease, but only incidentally, under definite conditions, as a symptom in the course of other diseases.—We must say exactly the same of the greater part at least of the convulsions which sometimes appear in pregnant, and particularly in parturient women, and occasionally even at the present time seem to lay claim to a special position under the name of Eclampsia gravidarum et parturientium. The conditions under which the convulsions of parturient women occur are very numerous and variable, and we shall have to show hereafter that certain cases of these convulsions should, in fact, represent a special disease, for which the name once in use may be retained. Others of them, however, in which the patients had albuminuria before the confinement, must undoubtedly be interpreted in the same way as the spasms just mentioned as occurring in the course of kidney diseases, *i. e.*, they form a link in the chain of symptoms of the latter, and not a separate disease.[1]

But we go a step further. In our opinion it is not even correct to keep to the special name of eclampsia for the convulsions with loss of consciousness which so often occur in children at the beginning of acute febrile diseases, as in pneumonia, erysipelas, and the acute general exanthemata, also as an introduction to poliomyelitis anterior acutissima atrophica (the spinal paralysis of children). Here, too, we have to deal with a symptom of another fundamental disease, not with a special affection; and on this account we may here refrain from a discussion of the question whether the fever as such, the elevated temperature, or a possible hyperæmia of the brain, or some other influence, furnishes the cause of the convulsions. It is possible, of course, that in some of these febrile convulsions the same processes are actually going on which characterize eclampsia (according to our assumption), but at present it is not proved.

Finally, there must also be distinguished from eclampsia the seizures which sometimes appear in severe chronic lead-poisoning. It has nothing to do with the matter whether the saturnine cerebral symptoms arise from the immediate action of the lead upon the nervous elements of the brain, or whether they are brought about through an intermediate action upon the vessels (anæmia)—they represent only one of the symptoms that result from the organic changes effected by the lead.

What is there now remaining of what was formerly recognized as eclampsia? And are we altogether justified in still retaining this name? We believe so, and are of opinion that the title of *eclampsia* should be reserved as the name of an *independent affection*, which, it is true, can at present only be defined by its clinical symptoms. We propose that *the designation eclampsia should be made use of for those cases of epileptiform spasms which—independently of positive organic diseases—present themselves as an independent acute malady, and in which*—so far as our present knowledge allows us to judge—*the same processes arise, generally in the way of reflex excitement, and the same mechanism in the establishment of the paroxysms comes into play, as in the epileptic seizure itself.* In this way, as we see, the designation of eclampsia as an acute epilepsy finds greater authorization; at the same time it is distinguished from true epilepsy by the lack of a persistent central change, which latter impresses upon epilepsy the character of a chronic condition. In the case of eclampsia, where this chronic change is absent, the manifestations, the seizures, disappear with the removal of the exciting irritation.

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[1] We can of course only touch upon this question here so far as it is related in a very general way to "eclampsia." For the particulars about eclampsia parturientium we must refer to diseases of the kidneys and of women.

Etiology.

Hughlings Jackson properly insists that only in extremely rare cases are simple peripheral irritations *alone* capable of exciting epileptiform convulsions or an eclamptic seizure, and that this is possible only if a certain physiological or pathological peculiarity on the part of the nervous system already exists. All observers are agreed as to this, that such a disposition to eclamptic seizures—such a convulsibility, as it is expressed—belongs particularly to early *childhood*. This is so striking that a special class has even been formed under the name of eclampsia infantum.

Upon what depends this great tendency of the nervous system in childhood to react upon peripheral sensory excitements, not only with local twitchings, but even with general convulsions and loss of consciousness, while they remain without any further results in grown persons? That this is not at all dependent upon an inherited neuropathic tendency, or at least only very exceptionally so, is evident from the fact that even on the most thorough investigation very often not the slightest trace of it can be discovered. Sometimes, to be sure, such an inherited disposition seems to exist, since at times we see all the children of a family affected with eclampsia. Moreover, it does not depend upon whether the children are weakly or strong, thin or fat, lively or apathetic. True, it seems that convulsions occur more readily in the presence of some morbid anomalies than otherwise, as especially in rachitis ; but it is very certain that such are absent more often than present. From the whole body of observations we should rather come to the conclusion that it is not due to definite pathological states, but that the physiological constitution of the central nervous system in children is so modified as to cause them, upon very slight irritation, to respond with spasms which may advance even to eclamptic seizures.

This conception is, in fact, quite generally recognized, and certain pathologists, as, *e. g.*, H. Jackson, even seek to give an explanation for it. This author, indeed, calls attention to the fact that the nervous system of children is, in the first place, still undeveloped, and, in the second, is undergoing development. Meanwhile this interpretation is one so very generally adopted that it scarcely contributes anything to the elucidation of our question.

In this connection we would refer to the well-known physiological fact that all reflex acts take place much more actively after removal of the cerebral hemispheres, which is also expressed by saying that the latter exercise an inhibitory action upon the occurrence of reflex acts. This reflex inhibitory action proceeding from the hemispheres, which was repeatedly ridiculed after the labors of Setschenow were made known, has been again rehabilitated, particularly by the experiments of Goltz.[1]

To render this reflex inhibitory action of the cerebral hemispheres intelligible, a voluntary suppression of reflex movements has generally been assumed. This undoubtedly sometimes happens, but Wernicke[2] has, in our opinion, correctly indicated another influence, namely, the passing over of every centripetal irritation, which takes place in a normal state of the nervous system, not only into the reflex paths, but also into the hemispheres. By this division a certain sum of the living force required in the irritation-process is claimed for the cerebrum; consequently the resulting movement must now be weaker, as, *cæteris paribus*, the reflected movement is proportional to the strength of the irritation which gives rise to it. Various circumstances, the more particular discussion of which in this place we must decline, only favor the view that in very early childhood the sum of the living force, which is required for the "accessory circuit" (Wernicke) in the cerebrum, is essentially less than in later years. For this reason the irritation passes much more vigorously to the reflex channels, and produces in them more powerful effects, *i. e.*, these very spasmodic symptoms.—We give the foregoing conception, with all reserve, as a hypothetical attempt at explanation of the unusual tendency to convulsions in early childhood which has been experimentally determined.

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[1] Functionen d. Nervencentren des Frosches. Berlin, 1869. pp. 39 et seq.

[2] Der aphasische Symptomencomplex. Breslau, 1874. p. 11.

Eclampsia in adults—in the sense of our former definition—occurs with disproportionate rarity. It is not possible to determine what is the nature of the conditions of the nervous system which predispose to it.

Only in a very general way observation teaches that *anæmic* and so-called *irritable* and *nervous* persons, even though they be robust, are the soonest attacked by eclamptic seizures; at least mention is made of these characteristics in most of the detailed histories of patients. Although we can explain these constitutional anomalies on neither physiological nor anatomical grounds, we must nevertheless retain them as clinical conceptions.

Exciting causes of the seizures. a. *In Childhood.*—Generally, although not always, a definite influence, an external irritant, can be proved to exist, with the operation of which the outbreak of the eclampsia coincides, and regarding the causal relation of which to the latter no doubt can be entertained, as after its removal complete recovery follows. The intensity of these irritations need not be particularly great; it may even happen that they are indicated by no other symptom, and that we are first led by the eclampsia itself to seek more thoroughly for their presence. By far the most frequently these sources of irritation involve the sensory nerves of the mucous membrane of the digestive tract from the mouth down to the rectum. The "teething convulsions" of children have always played an important part with mothers, and they actually do occur quite frequently, especially during the first dentition and when the eruption of the teeth is somewhat difficult. If these convulsions are more often observed during the cutting of the eye-teeth and molars than during that of the incisors, this may be for the reason that the latter, as a rule, come through more easily.—Equally well known are the "convulsions from worms." Very numerous instances are recorded in the literature of the subject, and every physician will be able to add to the number, where violent convulsions in children have been excited by the presence of round worms in the intestine, and have ceased with their expulsion. And here we may very properly remark how perfectly well the little patients were feeling up to the onset of the eclampsia, and how completely ignorant the parents are apt to be of the existence of the round worms, prior to the time of their discovery.—In other cases, again, indigestions furnish the exciting cause, or vomiting, or diarrhœa; and here also belong the oft-detailed instances when children have been visited with eclamptic paroxysms on taking the breast after the mother or nurse has undergone some violent mental change. We certainly need not here consider at length that these cases should not be confounded with those where children present the symptoms of hydrencephaloid as a consequence of profuse diarrhœas; here it is often very vigorous, well-nourished children which are seized with eclampsia at the commencement of fresh attacks of diarrhœa.—It is established by numerous examples that occasionally even irritants which affect other sensory nerves, *e. g.*, those of the external integument, may likewise provoke eclamptic seizures.

Experience, however, further teaches that eclampsia may be caused in other ways than through centripetal irritations. Thus it is sometimes observed after violent mental impressions like terror or excitement. Cases even occur in which not the slightest exciting cause can be made out.

b. *In Adults.*—As already mentioned, eclampsia is much more rarely observed in advanced age than in childhood. But here, too, external sources of irritation, sometimes of very insignificant intensity, play a prominent part among the inducing causes. Here, too, these may proceed from the most different sensory nerves, from the external skin (wounds[1] or burns), from the mucous membranes, especially that of the intestine (even in adults eclamptic seizures have been noticed as a consequence of tapeworms), from the genital organs, the ureters and gall-ducts on the passage of calculi, etc. We consider it unnecessary to reproduce the particular observations bearing on this.

To eclampsia parturientium only must we recur once more in this place. We have already remarked above that certainly most of the accidents to which this name is applied should be held to be entirely distinct from eclampsia, but that a few cases of convulsions in pregnant and parturient women must really be regarded as eclampsia. This is the fact with those cases where no albumen whatever is present, or merely a trace, after the paroxysms only, and probably then the result of the latter. The mode of origin of many of these eclamptic paroxysms, which occur in the period of gestation without kidney affections, is certainly very obscure, and we can only learn from further experiment in what way they act; but Wernich[2] has furnished an attempt to explain some of these cases, which seems to us deserving of attention. Wernich, namely, observed in some such patients pronounced symptoms referable to a considerable pressure upon the sciatic nerve from the enlarged uterus. His opinion now is that this lesion of the sciatic might act in a similar way as the injury of the same nerve in guinea-pigs, *i. e.*, that thereby a condition of increased irritability might be established in the pons and medulla oblongata. Then, if a further irritation in the region of some other peripheral nerve be added to it, particularly of the sensory nerves of the sexual organs (*intra partum*, etc.), the eclamptic seizures would break out, according to the analogy of the irritation of the epileptogenous zone in the guinea-pigs.—These cases would really then have to be considered as eclampsia parturientium (in our sense).

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[1] *Marowsky, e. g.*, communicates a very exceptional case of this kind. Deutsch. Arch. f. klin. Medicin. Vol. III.

[2] Berl. klin. Wochenschr. 1872. No. 42.

Even in adults the disease may, as in children, be called into existence on some rare occasions by mental influences.

From all that has previously been said, it is self-evident that we cannot speak of a special *pathological anatomy* of eclampsia. If patients die in the seizures, then the conditions found *post mortem* are either incidental and subsidiary, or they are connected with some inducing cause, or finally, they are direct results of the convulsions themselves. Definite changes of the central nervous system, which would be characteristic of eclampsia, are unknown.

Symptomatology.

In the description of the symptoms we may briefly sum up what we have to say as follows:

With *children* the disease either sets in suddenly, *i. e.*, the seizures constituting it overtake them in the midst of perfect health, or more or less marked premonitions precede its outbreak. These *warnings* may be of two kinds: they are either connected with the inducing causes, and consequently have not the least that is characteristic about them (diarrhœa, redness and painfulness of the gums, symptoms of worms), or they proceed from the nervous system itself. In regard to the latter there may be a great diversity; the following are most frequently observed: the children do not sleep quietly, often start up violently, but as if frightened; if they already have teeth, gritting of the teeth is not infrequently observed. In the waking state the little patients are often cross, wilful, and sullen. Besides the gritting of the teeth, other spasmodic contractions in single muscles or groups of muscles may also appear, most commonly in single muscles of the eye (strabismus and rolling of the globe), in the facial muscles (risus sardonicus), bending in of the fingers, extension or incurving of the feet.

Now, whether phenomena of this kind have gone before or not, the proper characteristic convulsions break out very suddenly. We need not here give any extended description of them, for these eclamptic paroxysms behave throughout exactly like the major epileptic attacks ; so it is sufficient simply to refer to these latter.—If an inducing cause is discoverable, and can be easily removed, the seizures also then generally disappear. If the removal of the etiological influences is impossible, the convulsions are repeated ; sometimes this happens even after doing away with the source of irritation. Thus the paroxysms may follow one after another on three, four, or five days ; between them the children either seem to be quite well, or they remain in a somnolent state, quite in analogy with the status epilepticus (*état de mal*). In this case the issue is generally fatal, the coma being, indeed, continuous. But even when the paroxysms are less frequent and the intervals greater, eclampsia is always a more dangerous affection in small children than in adults. It is true, the percentage of recoveries decidedly exceeds the fatal cases ; but the popular dread of the convulsions of small children has a real foundation so far as that they succumb during the attack itself to spasm of the glottis or venous hyperæmia of the brain much sooner than adults.

It must also be added that the partial muscular twitchings and the like, previously described as "precursors," are frequently observed under analogous etiological conditions, without true eclamptic paroxysms being associated with them.

In *adults* the attacks generally appear suddenly, if the inducing cause is actually in operation ; thus *intra partum*, after a wound, etc. Definite premonitions, which would be referable to the eclampsia itself, generally do not exist. Here, too, the paroxysms have a character as pronounced as in genuine epilepsy, so that a description is unnecessary.

Pathology.

We have previously expressed the opinion that we ought not to employ the designation eclampsia, as is now customary in practice, for all forms of spasm which occur occasionally and transiently under the form of epileptic seizures. We should rather understand by this term only such cases as are *similar to the true epileptic attacks*, not merely in their external clinical features, but *in the mechanism of their development.* It is hardly necessary for us to bring forward special proof that this resemblance does exist in the fullest degree in regard to the paroxysms which we have above indicated as "eclamptic." The identity of pathogenesis of our eclamptic attacks and of the proper epileptic ones is recognized in almost every quarter. Here, too, as in the major epileptic seizure, we have to deal with an excitation of the reflex centres situated in the pons and medulla oblongata (especially the vaso-motor and convulsion centres). For the same reasons as in the case of epilepsy, the study of the single cases necessarily leads to this conclusion.

But, as opposed to epilepsy, no persistent change in the parts of the brain mentioned exists in eclampsia. Consequently, the latter is also not chronic, but the disease ends with a few paroxysms following quickly after each other, or even with a single one. As another consequence, this disease scarcely ever breaks out spontaneously, but is almost always excited by a centripetal irritation.

Furthermore, in eclampsia a certain "predisposition" must also be assumed to exist; but, in our opinion, this is to be regarded otherwise than in the case of epilepsy, as in eclampsia we have to deal with conditions which are not limited to the pons and medulla oblongata alone, but either affect the whole nervous system, or in them perhaps the central portions mentioned may even be wholly intact (as in eclampsia infantum), or, finally, such, if limited possibly to the pons and medulla oblongata, as rapidly disappear again on the removal of the inducing cause (as perhaps in the form of eclampsia parturientium mentioned above).

Of the connection and the reciprocal relation which sometimes exist between eclamptic seizures and epilepsy, we have already treated *apropos* of the latter. The partial muscular twitchings, especially in children, often precede the proper eclamptic seizures, or even sometimes appear without the latter, notwithstanding the presence of causes that usually produce them. These twitchings should be judged of in the same way as the aura epileptica and the interparoxysmal twitchings which occur in epileptics.

Prognosis.

The prognosis, so far as a restoration to health is concerned, is favorable in eclampsia, since the inducing causes of it are generally accessible to treatment, and the general "predisposition" present may likewise either be to a certain extent combated (anæmia, nervousness), or corrects itself (the period of childhood). But, since the possibility of an epilepsy in process of development must always be borne in mind, the favorableness of the prognosis is liable to a certain limitation. Meantime the danger that permanent sequelæ, such as paralyses, contractures, or idiocy, may remain after the seizures, is to be feared in very exceptional cases only, because it is extremely rare for the paroxysms as such to occasion material intracranial lesions, and, on the other hand, the cases where such were already present, and had general convulsions associated with them as a symptom, certainly do not belong to eclampsia.

The seizure itself in an adult is subject to the same prognosis as in the case of epilepsy.[1] We have already expressed ourselves above about eclampsia in childhood.

Treatment.

The therapeutic measures must, if possible, be directed alike against the underlying tendency and against the inducing causes. In regard to the former, it frequently happens that no direct treatment is possible, especially in small children, and also in adults, if we have nothing definite to take hold of. If anæmic conditions are present, we must proceed against them according to well-known modes of cure. In the same way where there is great "nervousness or nervous irritability," the curative procedures which generally are of avail must be brought into use. (Compare the appropriate section of the present work.)

Every source of irritation, of which it may be assumed that it has excited the spasmodic seizure, must be removed by suitable remedies. Very frequently, on taking it away, the attacks vanish at once. These are the cases formerly so much spoken of as cures of epilepsy by anthelmintics, by calomel, etc. Of course, we need not here go into the details of the particular cases. The treatment will need to be very various, according to whether constipation or diarrhœa exists, whether helminthiasis is present, or the disturbance of a peripheral nerve, an acute indigestion, overloading of the stomach, or difficult dentition. It is denied in many quarters that in the last-mentioned case scarification of the gum is of the benefit claimed for it. I confess that I still employ this in teething children with eclamptic seizures, if I find the gum swollen and reddened in certain places where the eruption of a tooth is to be expected.

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[1] The paroxysms of parturient women cannot be judged of with reference to their danger, as the observations at present available consider all forms of spasm in these patients indiscriminately.

How are we to act in the presence of the seizure itself? In adults, on the same principles as in the real epileptic paroxysm; for the sake of brevity, we refer to the rules laid down when speaking of it. But in children also we should guard against officiousness; it will be necessary to interfere only when affairs take a threatening turn. Of course, we are not to expect that a spasm of the glottis already existing in the seizure itself can be averted by enemata with musk or assafœtida, or by mustard-plasters. Still, if a persistent coma, with symptoms of hyperæmia of the brain, has followed upon repeated attacks, after the analogy of the status epilepticus, we may try active measures of this kind, together with cold affusions to the head, and also, according to circumstances, irritating enemata or even local abstractions of blood. Oxide of zinc and the other "nervines" seem to exercise no noteworthy effect upon these eclamptic outbreaks which are rapidly repeated, or recur one after another on several days. Further observation alone can teach us what will be the action in this respect of bromide of potassium (in the form of enema) which has been so much praised of late.

TETANUS.

BAUER.

TETANUS.

The very copious bibliography of the subject will be found carefully collected in several monographs; especially the following:

W. Trnka de Krzowitz, Comment. de tetan. Vindob., 1777.—*A. S. Friedrich*, Diss. inaug. de tetan. traumat. Berlin, 1837.—*Blizard Curling*, A treat. on tetan. London, 1836.—*Funk*, Die Rückenmarksentzündung. Bamberg, 1849.—*J. Gimelle*, Mém. et observat. du tetanos. Journ. de Bruxelles. 24. Vol. 1857.—*Thamhayn*, in Schmidt's Jahrb. Bd. 112.

Manuals and Cyclopædias: *Rochoux*, Encyclopädie der med. Wissensch. nach d. Dict. de méd. frei bearb. von Schmidt und Meissner. Leipzig, 1833.—*J. Copland*, Dict. of med.—*C. Canstatt*, Handb. der med. Klinik. III. 1.—*M. H. Romberg*, Lehrb. der Nervenkrankh. I. 2.—*K. E. Hasse*, Krankh. des Nervensystems.—*M. Rosenthal*, Handb. der Nervenkrankh. Erlangen, 1870.—*Bednar*, Die Krankh. d. Neugeb. u. Säugl. Bd. II. Wien, 1851.—Handbuch der Chirurg. von Pitha u. Billroth.—Tetanus, von *E. Rose*, I. Bd. 2. Abth. 1. Heft. 1. Liefg.

Dazille, Observ. sur le tetanos. Paris, 1788.—*A. Colles*, On the cause of the disease termed trism. nasc. Hosp. Rep. Vol. I. Dubl. 1818.—*Finkh*, Ueber den sporadischen Starrkrampf d. Neugeborenen. Stuttg. 1825.—*R. Froriep*, Ueber die Ursachen des Wundstarrkrampfes. In Neue Not. aus dem Gebiete der Natur- und Heilkunde. No. 1. 1837.—*Larrey*, Mém. de chirurg. milit. et campagnes. Paris, 1812–1817.—*J. Morgan*, A lect. on tet. London, 1833.—*Lepelletier*, Rev. Méd. 1827. tom. IV.—*J. Schneider*, Vers. einer Abhandlung über den Kinnbackenkrampf neugeb. Kinder. Fulda, 1805.—*J. Swan*, An essay on tetanus. Lond. 1825.—*Schoeller*, Neue Zeitschr. für Geburtsk. Bd. V.—*Kniesling*, Deutsche Klinik. 1849. No. 7.—*v. Tscharner*, Ueber den Tetanus. Bern, 1841.—*v. Dusch*, Zeitschr. f. rat. Medic. 1852.—*Rokitansky*, Ueber die Bindegewebswucherung u. s. w. Sitzungsberichte d. Wiener Akad. 1857.—*H. Demme*, Zur path. Anatomie des Tet. 1859; and allg. Chirurgie der Kriegswunden. 1861; and Schweizer Zeitschr. II. 1864.—*Heiberg*, Norsk Mag. 1861.—*Wunderlich*, Archiv der Heilk. 1861, 1862, 1864, 1869.—*E. Güntz*, Beobacht. über Temp. u. s. w. Diss. Leipzig, 1862.—*Leyden*, Virch. Archiv. Bd. 26. 1863.—*L. Concato*, Sul tetano. Bologna, 1865.—*Billroth* u. *Fick*, Vierteljahrschr. der Züricher

naturforsch. Ges. VIII. 1863.—*Hutchinson*, Med. Tim. and Gaz. 1861, Apr.—*Richardson*, Brit. Med. Journ. 1859.—*Brown-Séquard*, Journ. d. Progr. des sc. méd. 9. Sept. 1859; and Bull. de Thér. 1861.—*Koenig*, Das Gesicht des Tet. Arch. der Heilk. 1871.—*Kussmaul*, Berl. klin. Wochenschr. 1871.—*Clifford Albutt*, Trans. of the path. soc. XXII.—*Michaud*, Arch. de phys. norm. et path. Nr. 1. 1872.—*Verneuil*, Gaz. des Hôpit. 1872.—*A. Vogel*, Deutsch. Arch. f. klin. Med. Bd. X.—*Lockhart-Clarke*, Med. chirurg. Transact. Vol. 48. 1865. —*Dickinson*, Ibid. Vol. 51. 1868.—*Heinecke*, Deutsche Zeitschr. f. Chirurg. Bd. I. S. 267.

Introduction.

A high degree of perfection was reached even in very early times in the mere description of tetanus; the explanation of the symptoms, however, followed all those changes through which scientific pathology has passed during the centuries.

Among the writers of antiquity, Aretæus has given the best and most concise description; he has also stated the causes of the disease, for the most part, in accordance with the views of the present day.

The causes of tetanus, according to him, are very numerous; it occurs especially after injuries, also after exposure to cold: hence most of the cases are encountered during the winter season. But the disease may also follow abortion, or a blow upon the neck. Women are more disposed to it than men; and of the different ages, childhood is especially liable to the affection, though in a less dangerous form.[1]

During the years which followed there were many who wrote concerning tetanus, and among these contributions may, in truth, be found numerous correct observations and sound views, though errors are also not wanting. But an essentially clearer insight into the nature of tetanus was not, indeed, obtained until surgery had risen to considerable importance, such as it attained during the time, and especially by the instrumentality, of Ambrose Paré; for, with surgery arose also an interest in this affection, which most frequently presents itself in the form of an accidental surgical disease.

With the introduction of firearms there was gradually developed a surgery of war—military surgery—and in the great number of battles rich opportunities were afforded the military surgeons to observe traumatic tetanus; in fact, most of the literature of the subject has been contributed by military surgeons. Further, the colonies of European nations in hot climates, where the disease is very common, furnished material for and developed an interest in, the study of tetanus.[1]

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[1] De causis et signis acut. lib.

The knowledge of tetanus was at first not directly favored by the great advancements made in pathological anatomy, but the development of physiology and experimental pathology has essentially advanced the knowledge of the pathology of tetanus.

By tetanus is denoted a form of disease in which numerous groups of muscles remain in a condition of *continuous tonic spasm;* the tonic contraction generally begins in the muscles which move the lower jaw, the œsophagus, and the neck, and spreads thence to the body, especially to the extensors of the vertebral column. The tonic spasm continues uninterruptedly, but generally paroxysmal exacerbations are observed, whereby also clonic convulsions may be produced, following which again partial relaxation may take place. These exacerbations frequently follow peripheral irritation; in a word, *the reflex-irritability is increased.* Accordingly, the term tetanus refers to a form of spasm, and the conception of the disease is a purely functional one, without determinate anatomical basis.

Tetanus, in general, is that condition of the muscles in which the separate motor impulses follow each other in such rapid succession that relaxation of the muscle is not possible, and it remains uninterruptedly in a state of contraction. Even in those cases in which the spasm seems to be continuous, it is evidently the result of separate centrifugal impulses, which, however, act upon the muscle as a unit, and keep it in a state of contraction. Tetanic contractions are therefore *tonic spasms;* they correspond to no anatomical unit, but are found in various irritative conditions.

Clinically the tonic muscular spasms are not pathognomonic of tetanus; they of themselves have no specific peculiarity, but their arrangement and succession give the disease its character. Therefore it is clearly desirable to draw the line of distinction as sharply as possible between tetanus, as it is characterized by its entire group of symptoms, and other similar processes.

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[1] Regarding the numerous essays on tetanus, consult *J. Gimelle*, l. c. pp. 12 and 13.

In tetanus the tonic muscular spasms do not affect all the muscles in the same degree; they always implicate certain groups of muscles more distinctly than others. In this way by the overpowering traction of certain muscles the body undergoes changes of external form. Formerly great value was attached to these changes of posture, and ever since Hippocrates these variations have been the foundation of the division of tetanus into different varieties. Much the most frequent is that in which, besides the locked jaw, the contraction of the long extensor muscles of the back and the muscles of the neck draws the head backwards, and bends the vertebral column into the shape of a bow with the convexity forwards; this was called *opisthotonos*. Then they also distinguished an *orthotonos*, when the body was stretched out straight, and an *emprosthotonos*, when the vertebral arch was directed backwards. When the closure of the mouth was alone prominent, the case was called *trismus*. To this original division Boenecken afterwards added another form, *pleurothotonos* (or tetanus lateralis, according to Sauvage), when, by the contraction of the muscles on one side, a lateral curvature of the body occurred. There was a protracted discussion concerning the last named variety, and its existence was denied by many. Quite recently E. Rose has not only denied the existence of pleurothotonos, but has also pronounced against the division according to the varying posture of the body, since he recognizes *opisthotonos* as the true and only form of tetanic muscular contraction. This much is certain, the frequency of opisthotonos so far preponderates that the other varieties proportionately disappear, and hence no division need be made. Most authors acknowledge that they have seen only opisthotonos, and appeal, for the other forms, to isolated meagre accounts of others. But it may very well be supposed that some delusion or mistake lies at the foundation of these isolated observations; this seems to be the case, according to E. Rose's criticism, in Larrey's reports, who even deduced the subsequent spasmodic position from the situation of the wound on the anterior or posterior part of the body.

There is also no ground for considering *trismus* as a special variety of tetanus, since such a division at best can correspond only to a quantitative difference. But the distinction is also not valid, because generally the muscles of the neck are also implicated, though perhaps only to a slight degree. Hence, trismus is a partial manifestation of tetanus, a synonym for locked-jaw.

Another classification is founded upon the course of the disease as regards its duration, an acute and a chronic variety being distinguished—the latter also called *tetanus mitis*. But this classification is also defective, as is proved by a consideration of the differences in the course of the disease at different times. Tetanus in very many cases ends fatally within a few days; but if convalescence sets in, it is gradual, the symptoms disappearing very slowly during the course of weeks. It is exceptional for death to occur at a late period. Such are the differences in the course of the symptoms. It is claimed that the acute and chronic forms likewise differ in their symptoms; but seeing that many cases of the so-called chronic or mild variety prove fatal within three or four days, *this* designation is at all events not very happily chosen. The best plan would be simply to lay stress upon the intensity and extent of the manifestations, and to drop every attempt at further divisions of the subject.

Etiology.

The knowledge of the etiology of a disease is but little promoted by selecting Latin terms to express the current views in regard to the causes which give rise to it, and attempting by this means to establish a classification. This is true of the early and pernicious etiological grouping of tetanus, which chiefly recognized four varieties: a traumatic, a rheumatic, an idiopathic, and a toxic tetanus. These designations, which are still in use, are to be rejected, if for no other reason, because they are in part given to diseases which are not at all related to tetanus—*e. g.*, to intermittent and hysterical toxic spasm.

The number of persons attacked with tetanus is small under any circumstances. In the greater number of patients the outbreak of tetanus is preceded by an *injury;* more rarely, in cases unaccompanied by any external injury, patients give a more or less distinct history of having *taken cold;* and in quite isolated instances there is a so-called *spontaneous development* of the disease, although in the latter class of cases different observers have assigned the most diverse causes to the individual attacks which have come under their notice.

Tetanus also appears in *new-born children*, and, indeed, within a short time after birth, as a rule within the first nine days of extra-uterine life. As this period of time is rarely overstepped, the disease must be considered as connected with the falling off of the umbilical cord. It is at present generally agreed that tetanus neonatorum is to be considered as of traumatic origin.

The numerical relation of tetanus to other diseases is not the same in all places, since in some districts, especially in *tropical* countries, this disease occurs much more frequently than in temperate and more frigid zones. At the same time it is true that there is a difference between different *races of men* living together in these tropical climates; the colored races being more subject to the affection than the Europeans living amongst them. The latter, however, are attacked more frequently than at home. Finally, in one and the same place there are variations in the frequency of tetanus at different times.

The frequency of tetanus in a temperate climate can be approximately seen from the following comparison:

In a period of thirty-two years 113,020 patients were received at Guy's Hospital, of which 72 had tetanus; therefore, one case of tetanus among 1,570 patients. In comparison with the whole number of deaths, the deaths from tetanus during the period from 1848 to 1853 in Guy's Hospital amounted on an average to 0.056 per cent., in London to 0.0025 per cent., in the whole of England to 0.0031 per cent.[1] In the Vienna general Hospital in the years 1855–64 among 239,911 patients there were 50 cases of tetanus; therefore one case to every 4,798 patients.[2]

E. Rose reports concerning Berlin, that in the Bethanien Hospital in the years 1847–64, 0.08 per cent. of all the patients died of tetanus, and in comparison with the total number of fatal cases the mortality from tetanus was 0.69 per cent. In the whole city, in the year 1867, of 683,673 inhabitants 275 died of tetanus, that is, 0.04 per cent. of the population, but of these 266 were new-born infants.[3]

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[1] See the comparison by *Thamhayn* in Schmidt's Jahrb. 1861. Bd. 112, and *A. Poland*, Guy's Hosp. Rep. Vol. III. p. 3.

[2] *M. Rosenthal.* Handb. der Nervenkrankh. p. 337.

[3] *E. Rose*, in Handb. der Chirurg. v. Pitha u. Billroth. I. Bd. 2 Abth. 1 Heft. 3. Liefg.

Besides demonstrating the rarity of the disease, these statistics with regard to certain districts in Europe also show differences in the frequency with which tetanus occurs in these various places. These differences become yet more marked if tetanus in newly born infants is considered. Curling states that in England the disease is very rare among the newly born; in Dublin, however, J. Clarke reports concerning his establishment that until the year 1782, 2,944 children among 17,650 died of this disease. Clarke sought the cause of this excessive mortality in the impurity of the air, and hence provided for purifying it. Afterwards, among 8,033 children only 419 died of this disease.[1] According to the report of Holland, the disease is usually rare in Iceland, while upon the neighboring island of Heimaey the population would die out if it were not recruited by immigration, since almost all the children die of tetanus. The disease prevails in the same way in St. Kilda, one of the islands off the west coast of Scotland.[2] In Stockholm, in the year 1834, Cederschjöld saw a very great mortality among the children in the lying-in asylum, as in six months about 40 died of tetanus.[3] In some parts of Germany, too, the disease is at times observed with greater frequency, as in Fulda, also in Hungary; while it is reported from Paris, Vienna, and St. Petersburgh that the newly born are rarely attacked with tetanus.

Many authors make statements in regard to the frequent occurrence of tetanus in tropical regions. Even Bontius gave this information in regard to India. The most unfavorable reports, especially during the eighteenth century, were made with regard to tropical America (Guyana and Cayenne) and the West India Islands. According to the accounts given, tetanus in these regions must be looked upon as a veritable public scourge, and it greatly injured the prosperity of the colonies, owing especially to the excessive mortality among negro children. According to Rainal, in Louisiana half of all the newly born negroes died, and also a large number of the white children; he found the Antilles equally as dangerous at this period of life.[4] Hancock makes the same statement in regard to the colonies at Essequebo and Demerara.[5] According to Fourcroy, at St. Domingo 80 out of every 100 negro children died before they were nine days old.[6] Barrère, Bajon, Poupée Desportes, Mosely, and others, have written to the same effect.

According to all these statements, the negroes suffer by far the most severely from the *mal de machoire*, the whites and Indians in a much less degree. Thus Campet writes that the disease appeared frequently among the negroes, but not among the whites.[7] Anderson remembered no case among the sailors at Trinidad during many years.[1] Dazille, who practised a very long time in the colonies, and had abundant experience with this disease, reports that among the white children tetanus was more rare; also that among an equal number of wounded a larger proportion of negroes than of whites were attacked with tetanus.[2] In tropical America the disease still prevails at the present time among the newly born, so that, according to Maxwell and Grier, a large number always die from this cause.[3]

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[1] Transact. of the Royal Irish Acad. Vol. III.

[2] See *Curling*, l. c.

[3] See *Friedrich*, l. c.

[4] Histoire philosophique.

[5] Edinb. Med. and Surg. Journ. Vol. XXXV.

[6] *Curling*, l. c.

[7] Trait. pract. des mal. des Pays chaud.

A numerical statement of the frequency of tetanus in the East Indies in recent times proves that there, too, it occurs much more frequently than with us. Peat, at Bombay, met with 195 cases of tetanus among 26,719 sick between the years 1845–51, or an average of 0.73 per cent; Morehead, during six years, found 0.8 per cent. In the years 1848–53, in Bombay, the proportion of fatal cases of tetanus was 3.9 per cent. of the whole number of deaths. According to Peat, among 11,929 natives 161 suffered from tetanus; among 2,733 Europeans, 21, a proportion of 1.3 per cent. and 0.77 per cent. Thus, a larger number of natives were attacked than of the Europeans.[4]

The reasons which may possibly lie at the foundation of these periodical and local variations in the frequency of tetanus will be more correctly appreciated when the conditions which contribute to the causation of the disease shall have been more accurately considered. It has already been mentioned that, in by far the greater proportion of the cases, tetanus occurs after a *previous injury*, so that a causative connection between the two must certainly exist. But since among the daily occurring injuries only a small fraction gives rise thereto, it is clear that the occurrence of traumatic tetanus must also depend upon other more special conditions. The question arises whether a part of these conditions is to be found in the *peculiar conditions of the wound.*

a. First of all, so far as relates to the nature of the wound, statistics show that the majority of cases of traumatic tetanus occur after lacerated and crushed wounds, with intrusion and presence of foreign bodies; therefore after gunshot wounds and burns. Yet the form of the injury is not absolutely important, and every variety of wound, howsoever it may be produced, may give rise to tetanus; its occurrence is, however, rare after clean cuts and stabs. Moreover, cases of tetanus occur which are clearly traumatic without any external injury, so that an internal lesion or concussion of certain centres of innervation must be presumed.[1]

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[1] Edinb. Med. chirurg. Trans. Vol. II.

[2] Observat. sur le tet.

[3] *E. Rose*, l. c.

[4] *Thamhayn*, l. c.

b. The *extent and severity* of the wound stands in no direct relation to the frequency of tetanus; for if extensive destruction by crushing, tearing, burning, compound and simple fractures, amputations, etc., may be followed by tetanus, yet it is much more frequently found after quite insignificant injuries, which are entirely disregarded by the patient, or quite escape notice.

c. The *seat of the wound*, the *region of the body* which has suffered, is a matter that exercises a decided influence in these cases; for wounds of the extremities are the ones most frequently followed by traumatic tetanus. Hence, by many, as by Hunter, Stafford, and others, special importance is ascribed to injuries of the tendons.

d. The *condition of the wound, its tendency to heal*, gives no sure criterion by which to judge of the liability to the disease. Some surgeons specially emphasize the fact that tetanus is frequently associated with wounds which subsequently lead to extensive necrosis in their neighborhood (Rose). It is furthermore stated that wounds with considerable swelling, with diffused redness and increased temperature, are often observed where tetanus sets in. This connection may, indeed, exist, but these factors can be found only in extensive wounds, while in the great majority of slight injuries the same result must be brought about by other causes.[2]

e. Tetanus may also occur when the wound has completely healed, and a cicatrix has formed. There is no limit to the time which may elapse between the injury and the outbreak of tetanus. The condition of the cicatrix may also vary; it may present no special peculiarity, while, in other cases, the nerves have been found distorted by its contraction, or foreign bodies have been found included in the cicatrix.

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[1] Cases of the latter sort are well known. Lately *P. Guttmann* has described such cases (Archiv f. Psych. u. Nervenkrankh. Bd. I. p. 730), in one of which, after death, no peripheral nor central injury could be found.—A short time since I saw a boy, in whom unmistakable and fatal tetanus occurred after a fall upon his neck without any peripheral wound.

[2] It must not be forgotten that the wound may first assume an unfavorable condition under the influence of tetanus, though, according to the assertion of *Travers* and others, the cicatrization of the wound frequently enough progresses without interruption.

According to the tendencies in the character and course of the wound certain conditions are indeed to be found which, by their frequent recurrence in tetanic affections, indicate a causative relation. But this influence can only be predisposing, their significance only that of accessory agencies, since they are often entirely wanting, and because also the nature of their influence upon the body varies greatly in different cases.

There is scarcely any injury, however severe or slight, which has not been the occasion of traumatic tetanus. The outbreak of tetanus shows indeed a certain analogy to metastatic pyæmia, since with both every possible kind of injury may under certain conditions give rise to the disease (*e. g.*, I saw a robust man in his twentieth year die of pyæmia after the extraction of a tooth skilfully performed). Pyæmia does not result from being wounded, but from the disintegration and carrying away of thrombi, and we must suppose the origin of tetanus to be somewhat similar.

Examples of the different varieties of injuries which have been followed by tetanus are found mentioned in rich profusion by Curling, E. Rose, ond others. It has been seen to follow the extraction of teeth, the application of cupping-glasses, the sting of a bee, or the catching of a fish-bone in the throat, and it has resulted from the application of a blister or the use of setons, or even from cutting the nails too closely. As an offset against these insignificant lesions, severe injuries and capital operations also in numerous cases appear as the cause. If the larger collections of cases be studied, a marked preponderance of the slighter injuries is observed —small lacerated wounds, injuries by splinters of wood, nails, etc. At the same time it is certainly to be remembered that these slight injuries occur with extreme frequency in ordinary life, and that the total number of such far exceeds the number of severe injuries.

The locality of the injury is given by Thamhayn in 395 cases, as follows:

Hand and finger	111	times, or	27.42	per cent.
Thigh and leg	97	"	25.08	"
Foot and toes	87	"	22.19	"
Head, face and neck	44	"	10.99	"
Arm and forearm	31	"	8.09	"
Trunk	25	"	6.28	"

According to Curling's table, in 128 cases the injury occurred 110 times on the extremities. But since the limbs are the most exposed to wounds, Curling has examined a table of 510 injuries with reference to this point, and found 317 of them occupying the limbs. Hence the proportion of injuries of the limbs in tetanus is greater than in wounds in general; *i, e.*, wounds of the extremities are more liable than others to give rise to tetanus.

Different views prevail as to the other conditions which, with the co-operation of the wound, cause the outbreak of tetanus. The assertion made by many, that every case of tetanus is traumatic, but the injury is often overlooked, needs no special refutation. A consideration of the relative frequency of wounds and of tetanus shows satisfactorily that some special cause must exist in the individual that is wounded in order to insure the development of the subsequent disease. It always has been and is still held by many that the cause is to be found in the fact of the person's "taking cold;" according to which theory the influence of external temperature on a wounded person, especially its rapid variation, acts as the immediate occasion of the outbreak of tetanus. By some observers the importance of the climatic and atmospheric influences is alone acknowledged, by others yet greater importance is ascribed to the unfavorable influences which may act upon the wound in the most various ways, to its improper treatment and irritation (E. Rose).

Military surgeons, under whose observations, indeed, most of the cases of tetanus come, have everywhere noticed that the frequency of tetanus among the wounded was evidently dependent upon the influence of the temperature. Thus, Larrey, surgeon-in-chief of the French army during the Egyptian campaign, saw the disease chiefly when the wounded were directly exposed to wind and storm and rapid changes of temperature.[1] Cullen considers the influence of cold moist air as an essential condition. Hennen states that, after the battle of El-Arich and after the capture of Jaffa, many wounded were attacked with tetanus when they lay in tents on the damp ground.[2] Huck mentions that after the battle of Ticonderoga nine of the wounded had tetanus after exposure to the cold during the night in open boats.[3] Schmucker noticed the occurrence of tetanus in Bohemia after the slightest wounds when cold nights followed hot days.[4]

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[1] Mém. de chirurg. militaire et campagnes.

[2] Principles of milit. surg.

[3] Med. observat. and inq. Vol. III.

[4] Chirurg. Wahrnehmungen. Bd. II.

The same opinion was held by Dazille, Chalmers, Bajon, Hillary, Dupuytren and others.[1]

What cold causes in many cases follows in others from other kinds of unfavorable influences, *e. g.*, from improper treatment of the wound. Lately E. Rose in particular has advanced this proposition, and supported it by arguments drawn partly from his own experience. It was the custom in St. Domingo to treat the negroes who had been flogged with sharply irritating embrocations to protect them from tetanus, from which many suffered. Dazille abolished this treatment, and with the best of results.

What Dazille accomplished among the negroes may be asserted in general terms to be true of the present age, viz., that there is a decrease in the frequency of tetanus among the wounded as compared with former times, and this fact is decidedly in favor of the view advanced by E. Rose; for, in the course of time, nothing has so much changed as the treatment of wounds in its widest sense. Dickson was one of the first to refer to the diminution in the frequency of this disease, and explained it by the improved methods of treatment.[2] There are, indeed, no comprehensive statistics in regard to the earlier marine and land wars which would allow of a direct comparison, but isolated data still permit an approximative judgment. G. Blanc states that of 810 wounded 30 were attacked with tetanus.[3] J. Lind saw 5 die of tetanus out of 6 cases of amputation,[4] and others have made similar reports. Modern history has only one such example to offer, namely, the battle at Lyon in the year 1834, where of 277 wounded 12 died of tetanus. On the other hand, the great wars of the present day, especially the American civil war and the last contest between Germany and France, show no such proportion of cases of tetanus among the great numbers of sick and wounded. (According to a report in regard to Werder's corps, among 24,262 sick, with 7,182 wounded, there were 45 cases of tetanus).

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[1] An interesting story concerning trismus neonatorum is told of a midwife whose sense of heat was diminished after a sickness, so that she prepared too hot baths for her little charges, and lost such an unusual number of children from trismus that the attention of the officials was attracted thereby (oral communication of *von Ziemssen*).

[2] Med. chir. trans. Vol. VII.

[3] Diseases of seamen.

[4] An essay on the most effect. means, etc.

It is consequently probable that very different influences may contribute to the occurrence of tetanus among the wounded; but necessarily the operation of so heterogeneous influences must agree in *one* point. Most of the facts tend to prove that the common result of these influences is a peculiar irritative condition of the peripheral nerves.

The facts which prove the origin of tetanus through centripetal conduction of disturbances in the peripheral nerves will be given later. In an etiological point of view the peripheral nervous irritation is the only point wherein the different causes agree. Certain examples point still more decidedly in this direction. Thus foreign bodies, especially when imbedded in nerve trunks (Hennen, Dupuytren, Béclard), are a special source of danger; so are also the tearing and twisting of nerves, as sometimes happens from the application of ligatures (Brodie, Dazille, and others), and hence certain operations that are attended with special liability to the accident (castration, for example). The same may likewise be said of ascending neuritis. From the analogy existing between these and other facts, we know that in all such conditions the disturbances are less likely to be due to positive interruption of conduction and destruction of nerve-fibres than to wounding of the nerves with retention of their conductive power.

The etiological conditions already mentioned hold true also in the tetanus, or, as it is usually called, the trismus of the new-born. The condition of the wound at the umbilicus, like that of wounds in general, shows no definite and regular changes. In most cases it is perfectly healthy.

Tetanus occurs also in connection with parturition or after abortion; but this is exceedingly rare. Here too we have conditions which may be considered similar to a wound.

Many cases of so-called spontaneous tetanus may perhaps be regarded, etiologically speaking, as belonging to traumatic tetanus, since in persons thus affected diseased processes have been found in the interior of the body which may have given rise to a state of irritation in the peripheral nerves of internal organs. The variations in this respect shown by different cases are almost as numerous as the cases themselves, and it is of course generally impossible to establish the real causative connection between these conditions and tetanus.

Intestinal worms are more frequently accused of being the cause of tetanus than anything else, and the proof is also derived *ex juvantibus*. But tetanus has likewise been observed in many other diseases. Bright saw it in acute rheumatism, with pleurisy and pericarditis; Rosenthal, in ulceration of the rectum. I have myself seen a young man die of tetanus which appeared during an attack of pleurisy with exudation. If such conditions as the irritation of worms are sufficient to produce tetanus, it follows, as a matter of course, that the cause of the affection may often escape observation even on a post-mortem examination.

Concerning the relation between tetanus and the fact of having taken cold no more can be said than with regard to the origin of other diseases that result from catching cold. The statements of patients on this point are often very decided: a rapid change of temperature accompanied with a severe chill, staying in a moist room, sleeping on the damp ground, being wet through while in a perspiration,—these are often the starting-points for the disease.

Some points still remain to be mentioned in the etiology of tetanus in general, which, however, contribute but little to the understanding of the nature of its origin. Thus statistics show an excess of males over females; youth and middle age are also numerically more severely taxed than advanced age. So, too, the robust and muscular are said to be more frequently attacked than the feeble.

Friedrich's tables give 252 cases of traumatic origin, 210 of which were males and 42 females; Curling found 112 males and 16 females. According to the collection made by Thamhayn, which comprised the statistics of Poland, of Lawrie, and other reports from Glasgow, and 110 other cases, 329 occurred among males and 68 among females. Yet these numbers do not give the true proportion, since in traumatic tetanus, to which most of these cases belong, the greater liability to injury among men must be considered.[1] The statements in regard to new-born children vary.

If the newly born are left out of consideration, the statistics of Friedrich show that the greater proportion of those attacked are between fifteen and twenty years of age; according to Thamhayn they are between ten and thirty.

According to the testimony of many physicians the mental condition has also an influence upon the occurrence of tetanus.

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[1] See, in regard to this, among other references, Med. Times and Gaz., 1854. Vol. XXIX. p. 376.

Thus military surgeons have noticed the disease more frequently among the conquered than among the victors. Lately, Erichsen in particular has emphasized the importance of psychical impressions; it is claimed that severe fright alone may be sufficient to excite an attack.[1]

A very large number of cases of poisoning are characterized by the occurrence of muscular spasms; yet the nature and succession of these spasms vary much with different poisons. The prototype of an agent which may produce spasms is strychnia. By this poison the extensors are especially thrown into tetanic contractions, and there is opisthotonos. Hence, the tetanic spasms of strychnia are clinically very similar to tetanus, and, in dividing this disease according to its etiological factors, a variety called toxic tetanus has been introduced, and strychnine poisoning has often been regarded as the type of this form.

The cause of this strychnine spasm is to be found in an abnormally increased irritability of the gray substance of the spinal cord, so that very slight irritations, which would otherwise have no effect, are able to excite severe general reflex motions. The arteries are contracted, the blood-pressure is increased by irritation of the vaso-motor centres. The irritability of the peripheral nerves is not thereby increased, the irritability of the sensitive nerves seems even to be diminished.

Brucine is very nearly allied to strychnia in its effect; but it is somewhat weaker in its action. An alkaloid is also found in opium, which is capable of exciting very severe spasms, namely, thebain. Ergotismus spasmodicus is characterized, in severe cases, by painful contractions of the flexors or by general tetanus, besides other symptoms; hence, ergotin belongs to the group of tetanizing agents. Picrotoxin and coffein also possess similar peculiarities. This is by no means a complete enumeration of the poisons which exert a tetanizing action; several others have this influence.

Tetanus occurs also in animals under the same conditions under which it occurs in men, especially in horses, goats, and sheep. There are periodical and local variations in the frequency of tetanus in animals also; traumatic tetanus is likewise the most frequent variety, and is especially common after castration.

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[1] *Pissling*, Virch. Jahrb. 1872. p. 82.

General Description of the Disease.

Tetanus, as a rule, begins with mild symptoms which gradually increase in severity to the maximum. The patients feel at first a sense of stiffness in the neck, together with diminished mobility of the lower jaw and tongue; deglutition is difficult. There is no constitutional disturbance, only a painful tension is felt in the above-named group of muscles. Then an increase in the symptoms follows at a slower or more rapid rate; the stiffness becomes more marked and extends to the muscles of the back and of the abdomen; finally, all the muscles of the trunk may be implicated. The preponderance in the action of the long extensors of the spinal column is so great that the back is bent with its convexity forwards, while the head is drawn back. The facial muscles of expression also become stiff, and this gives to the features a characteristic appearance. Finally, the muscles of the extremities may be affected with the spasms; only the forearms and hands frequently, according to some authors always, remain unaffected, at least among adults. The succession in which the separate groups of muscles are attacked, therefore, is in a *descending* order. The cramp gives the contracted muscles a board-like hardness, and opposes considerable resistance to voluntary motions and also to passive motions, because of the impossibility of causing relaxation of the antagonists. Sometimes the whole body can be raised up by the head, like a lifeless pillar. The power of swallowing is very much interfered with, and the voice is generally altered.

The consciousness, the intelligence, is not disturbed, except in a few cases just before death; but generally there is complete sleeplessness. The tonic spasms are continuous; they extend from above downwards and increase in intensity. Death may soon end the scene without the development of other symptoms. E. Rose has especially described this course as typical of acute tetanus. Yet this picture is not the rule; much more frequently paroxysmal exacerbations of the tonic spasms are seen, which last some minutes and then give place again to the previous rigidity. These attacks, in which the muscles contract to their utmost limit with lightning rapidity, give the body the severest shocks; they occur spontaneously or upon any intended motion as well as upon the most insignificant irritation applied to the periphery and the nerves of special sense. Of course, the reflex irritability is increased to a high degree. These shocks may follow one another in rapid succession so that the character of clonic spasms is assumed. The frequency with which these paroxysms are repeated, and this may be every half hour or oftener, is of great consequence in estimating the severity of the case; for a large number of patients die during the paroxysms. With the increase of the tonic spasms the constitutional symptoms become more serious, for by the rigidity of the thoracic muscles the respiration is interfered with, and the descent of the diaphragm is hindered by the contraction of the abdominal muscles. The patients experience a sensation of oppression and anxiety, they complain of pressure in the pit of the stomach, they are restless, and from the curvature of the vertebral column can find no easy posture in which to lie. The muscular contraction generally causes severe cramp-like pains, and the tormenting thirst, and frequently also the hunger, which cannot be satisfied on account of the locked jaw, join to make the whole condition of the patient one of terrible torment.

Micturition is either normal or there is retention of urine, which is secreted in diminished quantity; involuntary urination is very rare. Evacuation of the bowels is always very sluggish.

The skin is generally covered with profuse perspiration. The temperature, as a rule, is normal in this disease, or but slightly raised; it may, however, be enormously high, though this is generally the case only just before death.

As a rule, in the beginning, and often also in the further course of the affection, the pulse is only moderately accelerated to 80–90 beats; after longer duration it rises to 100, or even to 120; but just before death, with the increase of the temperature, the frequency is much greater, rising even to 180.

At this stage a slight delirium may also appear.

By the paroxysms of spasms the sufferings of the patient are all greatly increased; the pain grows more severe in proportion to the amount of contraction; the dyspnœa becomes so great as to threaten suffocation. This is said to occur from spasmodic closure of the glottis, as well as from spasm of the diaphragm. But, in my opinion, the immediate danger of the dyspnœa, as causing a fatal termination, has been exaggerated, since the narcotism from carbonic acid causes a relaxation of the spasms, and hence a diminution of the danger to life. The patients become cyanotic, to be sure, but the sudden occurrence of death certainly depends in most cases upon stopping of the heart.

In by far the majority of cases the disease proves fatal: from the rapidly increasing muscular stiffness or from the attacks of spasms, generally in consequence of the cardiac paralysis. Finally, the fatal termination may occur with the characteristics of weakness and exhaustion caused by the frequent spasms and the consequent difficulty of propelling the blood through the lungs, and gradual weakening of the circulation; or the spasms relax, there is increased rapidity of the pulse and rise of temperature, and the patient dies thus. The fatal termination occurs generally within the first five or six days, or even sooner—rarely later; yet cases are known where death from exhaustion has occurred as late as the twentieth day. The establishment of convalescence is always gradual, generally occurring in cases where the greatest severity of symptoms has not been reached; there is then a successive abatement of the attacks, and also of the tonic spasms. Many weeks or more may elapse before complete return to health. Sometimes the patients feel themselves to have entirely recovered, only a stiffness of the muscles and difficulty in moving still remains some time longer. Severe subsequent disturbance, deformities and curvatures from persistent contractions are indeed described, but it is at least questionable whether these belong to tetanus.

In children also tetanus may consist exclusively of a tonic spasm, without clonic convulsions. The spasm begins as trismus, and, indeed, in many cases it remains limited to the locked-jaw; hence the designation *trismus* has been specially employed therefor. Even before the disease is fully established, the children are restless, cry out often, and frequently lay hold of the breast, to let it go again. To the tonic spasms, which extend downward, but also implicate the extremities more frequently than with adults, may be united, as in adults, the severest paroxysms, which are brought on in part by insignificant peripheral irritations, because of the increased reflex irritability. Among nurslings the disease leads rapidly to collapse, signs of which are quickly seen. Death may occur in a few hours; it commonly takes place on the second or third day. The fatal termination is very seldom delayed, and recovery is yet more uncommon, even when the spasms are limited in extent or amount simply to trismus.

Pathological Anatomy.

Externally the dead body shows no peculiar changes. Usually rigor mortis sets in rather soon after death, depending doubtless upon the strong acid reaction in the previously active muscles. According to Curling's statement, the rigor is more persistent and more fully developed than is ordinarily the case. Although the rigor mortis occurs early, yet the muscles which were contracted during life relax first, for the cause which excited the tonic spasms ceases to act with death, and has nothing in common with the rigor mortis.

According to the observations of Nysten, the rigor generally sets in many hours after death, after the spasm has previously entirely disappeared; this statement is also confirmed by Wedemeyer, Wunderlich, and others. Sommer maintains that he has seen the spasm of the muscles of the jaw pass immediately into rigor mortis.

For a long time changes have been sought in the central nervous system of persons who have died with tetanus. In earlier records, too, many positive results are found, especially concerning the condition of the spinal cord. J. Gimelle gives an exhaustive résumé of the results of autopsies. Yet most of the changes mentioned consist chiefly in anomalies in the blood supply, hyperæmia of the cord and of its membranes. Similar reports are made concerning the changes in the cranial cavity. Sometimes extravasated blood and collections of serum were also found.

These and similar results were considered by many physicians sufficient to give tetanus a place among inflammatory diseases, especially of the central conducting apparatus. In individual cases unmistakable signs of inflammation and softening were indeed found whereby the inflammatory nature of tetanus seemed to be established beyond a doubt. But it was afterwards evident that in some of these positive reports other pathological processes had been confounded with the conditions above named (Funk); then, again, the many negative results given could not be ignored (Benj. Brodie, Ollivier, and others, although Ollivier declared in favor of the existence of spinal meningitis); and finally, the statements with regard to the existence of hyperæmia of the cord and its membranes had to be referred in part to post-mortem suggillation, since the precaution has not always been taken to lay the body upon its abdomen. Hence the question still remained open when Rokitansky described a proliferation of connective tissue in the form of embryonic tissue as being a constant condition in tetanus. Rokitansky found this foreign gray substance scattered through the white substance in the cord. The bulk of the cord was increased; it swelled up and bulged out more than usual on section, and had a firm elastic consistency. The principal microscopical changes were an increase of nuclei and destruction of the medullary substance; here and there agglomerations of fat granules were found, and a few amyloid bodies. The substance in question is found after death in a number of diseased conditions, and may finally change into fibrous connective tissue or into indurated tissue; but in view of the rapid and extensive destruction in tetanus it does not reach this point.[1] Rokitansky found a similar change in the peripheral nerves.

This discovery of Rokitansky was fully confirmed by H. Demme in many cases; the increase of connective tissue was furthermore recognized by E. Wagner in Wunderlich's cases, and in Fechner's cases the same changes were found in the anterior and lateral columns. The statements of Rokitansky have not received any further confirmation. Leyden has proved that the changes found by Rokitansky and Demme are not constant, and in part depend upon deceptive appearances, the result of imperfect methods of examination.[1]

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[1] Sitzungsberichte d. Wiener Akad. 1857. Bd. XXIV.

More recently, in addition to many negative results of post-mortem examinations, some few observations have been made public in which demonstrable changes had taken place. Lockhart Clarke, in particular, has described such changes; besides great hyperæmia, he found centres of softening in the gray substance, and also in the white substance. Yet he did not consider that the group of tetanic symptoms depended upon these changes.[2] Dickinson has likewise described masses of exudation found in the gray and white substance, by which a destruction and crowding aside of the tissues was produced.[3] Michaud also found hyperæmia and dilatation of the vessels, and an abundant increase of nuclei.[4] Benedikt found in one case hyperæmia of the spinal cord, especially about the cells of the anterior cornua, and granular degeneration of the cells, but no increase of nuclei.[5] Notwithstanding these positive results, those cases must also be taken into account in which careful examination shows no such changes. The only change usually met with is increased fulness of the vessels.[6]

With regard to the condition of the peripheral nerves, especially in traumatic tetanus, the older reports state that marked changes were frequently to be seen at the seat of the wound. Foreign bodies were found imbedded in the nerve-trunks; the nerves were crushed and torn; there was also inflammation and thickening, but always confined to the vicinity of the injury. The observations of Lepelletier were hence of importance, he being the first to describe the occurrence of an ascending neuritis in one who died from tetanus.[7] Then Froriep in several cases found red spots and varicose swellings of the nerves, alternating with parts which remained healthy, and this change extended from the seat of the injury to the spinal cord.[1] Similar changes were mentioned by Curling. This condition of neuritis ascendens, though not constant (Hasse), has yet been found repeatedly, and is of great value in the explanation of the whole process.

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[1] Virchow's Archiv. Bd. XXVI. 1863.

[2] Med. chirurg. Trans. Vol. XLVIII. 1865.

[3] Ibid. Vol. LI. 1868.

[4] Arch. de phys. normal et path. 1872. and *Friedreich*, Die progressive Muskelatrophie.

[5] Nervenpath. und Elektroth. 1874. p. 270.

[6] In nine cases which he examined, *E. Rose* found an increase of the weight of the brain above the average, which, however, was only a secondary phenomenon.

[7] Revue Méd. 1827. tom. IV. and in *Curling*, l. c.

Changes of an inflammatory character in the sympathetic nerve have been mentioned by some observers, as by Aronssohn, Dupuy and Andral. Swan considered these changes in the sympathetic, especially in the cervical and the semilunar ganglia, as being of great value in explanation of the nature of the disease, but the existence of these changes has not been further confirmed.

The *voluntary muscles* have generally a pale color; frequently rupture of individual bundles and extravasation of blood can be seen, and, indeed, this rupture of the muscles may be very extensive. More frequently, still, these lacerations are only microscopic. In single cases fatty degeneration of the muscles has also been recorded.[2] (According to J. Ranke the amount of fat contained in the muscles increases during tetanus.)

The statement of Desportes that the thighs were sometimes fractured by the power of the muscular action is rather incredible.

For a long time the greatest attention was paid to the question whether the heart is affected in tetanus. In a case observed by Howship, at the autopsy, eleven hours after death, the heart was found extremely contracted. Watson also saw the heart strongly contracted and hard as cartilage. No doubt, these conditions depended upon rigor mortis. In one case of strychnia poisoning Rosenthal found numerous ruptures of the muscular fibres and small extravasations. This circumstance merits no special discussion.

The changes in the internal organs are not always constant, and are either accidental or secondary. Thus there may be hypostasis and œdema in the lungs, or ecchymosis in the pleura. The liver is generally described as anæmic, the spleen as small and containing little blood. The intestines are normally distended or greatly contracted, in some cases hyperæmic (M'Arthur). The kidneys either show no change or they are overfilled with blood, cyanotic. Parenchymatous changes in the kidneys are more rare. Griesinger found them once, and they must be supposed to have existed in those cases of the disease observed by Kussmaul. The pharynx and œsophagus were found greatly contracted by Larrey, and the mucous membrane of a lively red.

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[1] Neue Notizen. 1837. Bd. I.

[2] The changes in certain organs known to be produced by an elevation of the temperature of the body are rarely found in tetanus, because the high temperature is usually only of short duration just before death.

In newly born children, besides many negative cases, there have been noted more frequently than in adults serous transudation into the vertebral canal, over-distention of the blood-vessels of the membranes and extravasations of blood into the brain and cord; but the same weight only is to be attached to these changes as has been mentioned above. The umbilical wound has been found changed in various ways, but often enough there has been nothing noticed at this spot, or the changes were exactly like those found on perfectly healthy children. Dr. Schoeller frequently noticed an inflammation of the umbilical arteries. In some cases phlebitis is also found, or inflammation of the peritoneum in the vicinity of the umbilicus.

Pathogenesis.

The symptoms which are found in a case of tetanus point to a disease of the central nervous system; but it is inadmissible to consider tetanus as an *inflammation* of the spinal cord, as was formerly done; the anatomical changes of the cord do not support such a view, and they are also too inconstant. Neither can the existence of a degenerative process, with proliferation of connective tissue, in the sense indicated by Rokitansky, be proved; this anatomical explanation appeared, from the beginning, to be insufficient to account for the symptoms, since the anatomical changes correspond to no simple form of disease, but the same changes are found in connection with very different diseased conditions (Demme, Wunderlich).

The lack of a satisfactory anatomical basis on the one hand, and, on the other, the manifold peculiarities in the course and the origin of tetanus, have repeatedly given rise to the supposition that this disease is the result of infection. Among others, Heiberg has sought to establish this theory; he declares that tetanus is a blood-poisoning in which the tonic spasms are excited by changes in the muscles themselves, while only the clonic spasms are caused by reflex action.[1] This hypothesis, however, is so arbitrary, and is in opposition to so many facts, that it may be dismissed without further consideration.

As a more accurate knowledge of reflex manifestations was obtained it was seen that tetanus depended on *reflex action*, the muscular spasm being reflex. This view was supported especially by Romberg, who looked upon the increase of the *reflex irritability* as the chief element in tetanus, this increased irritability being gradually developed under the influence of the peripheral irritation. According to this view a typical example of the combination of tetanic symptoms was found in the tetanus due to strychnia poisoning, which does indeed depend upon an exaggerated irritability of the gray substance of the spinal cord. This explanation is doubtless sufficient for certain forms of toxical spasms, especially that due to strychnia poisoning; yet the influence of other agents which give rise to spasms, as nicotin and pikrotoxin, is probably exerted directly upon the nervous centres which cause the spasms.

But the clinical symptoms of tetanus do not fully agree with those of strychnia poisoning. The muscular spasms in essential tetanus are at first continuously tonic in the muscles of mastication and generally also in those of the neck; gradually they increase in intensity and extend downward, but there are no paroxysms.

According to the purely reflex theory the phenomena in tetanus must be connected in the following way: an irritation from the periphery is carried inward in an unbroken, regular current, whence it follows, according to Pflueger's law, that in the medulla oblongata the transmission of the motion is made to such motor tracts as have their origin therein (muscles of the jaw, of the tongue, of the œsophagus). At first the conduction of irritation follows only the primarily affected (injured) nerve-fibres, because the etiological conditions of tetanus cause abnormal irritability of these. Later, the reflex irritability *in general* is exaggerated, so that the irritation of any sensitive fibre whatever excites general reflex spasms. According to this view the increased reflex irritability is an abnormal function which, under the stipulated conditions, occurs without any primary anatomical change.

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[1] Norsk Mag. XV. and *Thamhayn*, l. c.

It is easily seen that in this sense the reflex theory cannot be unconditionally accepted. Thus, those cases of tetanus which follow a wound of the neck or of the occipital region, the connection of which with tetanus cannot well be denied, are scarcely to be explained otherwise than by direct irritation of the centres controlling the spasm. It cannot be easily proved whether in other cases of tetanus a direct and not a reflex irritation may also occur. Different opinions may be held as to the nature of this irritation. It is possible that the peripheral irritation acts upon the vaso-motor system of the central motor organs, so that the spasms occur in consequence of reflex disturbance of the circulation.[1] The disturbance of the circulation may lead to further pathological consequences, especially to exaggerated reflex irritability which may arise sooner or later as a purely spinal symptom in the course of tetanus. But it is also possible that there may be a progressive extension of the diseased process from the affected peripheral nerve towards the centre,[2] which acts directly upon that as an irritation, and the presence of ascending neuritis in some cases seems to give support to this view. Anatomical changes may thus follow which are, at least partially, beyond control, but which need by no means always be the same.

The last supposition is supported by marked analogies, and if these are followed out more fully, the conclusion is reached that tetanus is first of all only a symptom which may appear under many different conditions.

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[1] See *Benedikt*, Nervenpath. u. Elektroth. 1874 p. 368, and *Heinecke*, Deutsche Zeitschr. f. Chirurg Bd. I.

[2] See, among others, *E. Hitzig*, Unters. über das Gehirn p. 192.

Analysis of the Symptoms.

The time which elapses between the occurrence of an injury and the outbreak of the tetanic symptoms varies greatly; generally it is from five to ten days, but the spasms may begin after a few hours, and, on the other hand, weeks may intervene. In rheumatic cases the fact of having taken cold is generally recognized but a short time before the beginning of the symptoms. Among the new-born the disease usually appears from four to eight days after birth, but may be delayed longer, until the fourteenth day.

Groetzner mentions a patient, who underwent amputation, who was attacked with tetanus the moment the crural nerve was included in a ligature, and died after six hours.[1] A very marked example of the rapid outbreak of the disease is found in the frequently quoted case of Robison: a negro wounded himself in the finger with a piece of porcelain, and after half an hour was attacked with tetanus. On the other hand, very long intervals are also known to occur; Ward, of Manchester, reports a case where the symptoms first appeared ten weeks after the injury.[2] According to Friedrich's investigations, in one case three months intervened. Morgan saw one case wherein tetanus appeared two months after the wound healed; the autopsy showed that a splinter of wood was included in the cicatrix.[3]

The first symptoms are generally almost imperceptible, the very earliest changes frequently being noticed immediately after awaking from sleep. Yet undefined disturbances of the general health, and pains similar to rheumatism, which are hard to define, may also be noticed some time previously. Renewed sensitiveness of the wound, with shooting pains radiating therefrom, may also be included in the list of these so-called prodromata. The commencement is occasionally, though rarely, acute, with a single marked chill.

The spasms.—The tonic spasms usually begin in the muscles of the lower jaw and œsophagus, and extend downward to other muscles; sometimes the attention is first excited on protruding the tongue. At first the lower jaw can still be separated from the upper; swallowing, even chewing, can still be performed; only a stiffness and tension is noticed in the neck. Patients with these symptoms may still remain out of bed for some days in tolerably good condition. But the increase and extension of the symptoms may also be very rapid, so that in a few hours the disease is fully developed.

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[1] Der Krampf, insbesondere der Wundstarrkrampf. Breslau, 1828.

[2] According to *Curling*, l. c.

[3] A lecture on tetanus.

It is stated that, as an exceptional form of the disorder, the spasms may first appear in the injured part and extend to others; while the muscles there are contracted and from time to time thrown into convulsions.[1]

As the disease progresses the jaws are more fully closed. Sometimes the two rows of teeth are firmly pressed against each other, the spasm of the œsophagus also increases, so that the introduction of even small quantities of fluid is rendered extremely difficult and fatiguing. The locked jaw cannot be opened by any power that can be used. Speech is difficult, and the voice is altered from the difficulty of moving the tongue, and also because the muscles of the larynx are implicated. As the facial muscles of expression are affected the expression becomes quite characteristic; the forehead is wrinkled, the eyebrows are drawn upward as when one is fatigued, the eyes are staring and motionless; the pupils are generally contracted, but there is rarely strabismus; the mouth is drawn out laterally, the corners are drawn down and the teeth are partially uncovered by the lips; the naso-labial folds are strongly marked. In this way the expression of the face is greatly altered, and there is seen mirrored in it a singular mingling of opposite feelings.[2] The patients look unusually anxious and frightened, but also, at the same time, wildly excited, and the mouth is in a half-laughing, half-crying position; hence the name "risus sardonicus." The altered features of the pale countenance cause the patients to appear older. Thus, Farr states that a man twenty-six years old was taken for sixty. The stiffness of the neck, slight at first, increases until the head is firmly fixed backward. The statement of Curling that in this way the so-called nutator capitis may change its action and become an extensor muscle, depends upon a false idea of the action of the sterno-cleido-mastoid. The long extensor muscles along the vertebral column bend the back with its convexity forward, the chest arches forward strongly, so that it seems very broad, and the body may finally rest only on the occiput and sacrum. The epigastrium is sunken in, the abdomen flattened, and, like all the contracted muscles, gives a characteristic sensation of hardness to the touch. In this way that spasmodic position of the body is brought about, which according to old custom is termed opisthotonos.

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[1] *Dupuytren*, Trait. théor. et prat. des bless. par armes de guerre; and *Key*, Guy's hosp. rep. Vol. V.

[2] Of course, this mingling of expressions arises from an undue preponderance in the power of certain muscles during a general spasmodic contraction of all the muscles supplied by the facial and trifacial nerves.—Consult *Koenig*, l. c.

The muscles of the extremities are more rarely affected by tonic spasms, those of the legs being somewhat more liable thereto, while, according to E. Rose, the forearms and hands invariably escape; a statement also made by other observers. But this is certainly a rule with many exceptions, as is easily shown in some cases by trying to perform passive motions with the forearm, when the increased resistance of the contracted muscles will be perceived. In the extremities, too, the position of extension is the one usually assumed, yet in single joints there may be flexion. Finally, it may be mentioned that spasmodic erection also occurs. The tonic spasms may vary in the different cases in extent and severity, yet they are characteristic of tetanus. In some cases the spasm consists from the beginning to the end in a persistent rigidity of the muscles, but in most cases there are paroxysms in which the muscular contraction is suddenly increased, to remain for a while in that condition, and then again to yield to the previous comparatively relaxed condition. This sudden and universal muscular contraction throws the body into severe spasm. From the synchronous traction of antagonistic muscles there is generally no marked locomotion of the different parts of the body, the opisthotonos becomes more marked, the head is pressed backward into the pillow, the body is thrown forward into a bow, and the limbs are stretched out with a severe shuddering of the whole body. The tongue may be caught between the teeth and be severely bitten. The attacks may follow each other so rapidly that the spasms may have more of a clonic character. Trembling of the muscles seems to be rare, though Wunderlich and Kussmaul noticed it. During the paroxysm the patient becomes cyanotic, and is tormented with a sense of suffocation from the contraction of the pharynx and glottis, and a feeling of mortal dread; he foams at the mouth, and his chest seems as if wedged between unyielding pillars. The frequency with which these attacks recur varies in different cases. It is also less at the commencement of the disease, increases during its course, and finally, in cases which end favorably, or towards the fatal termination, decreases again. Sometimes the attacks are more frequent during the night than during the day. Thus the free intervals vary from ten minutes to hours. The duration of the spasmodic attack also varies; as a rule one or two shocks run through the body and then there is rest again, but the severest spasms may last many minutes, indeed, with slight remission, even hours.

The attacks recur in part spontaneously, without any recognizable external cause, in part from the most insignificant irritation acting upon the nerves of sensation, or the organs of the higher senses. Frequently a proposed voluntary motion gives rise to the spasmodic action, and this is especially the case with efforts at swallowing, so that the disease bears a resemblance to hydrophobia.[1] The peripheral impressions which give rise to general spasms are frequently extremely insignificant; the rustling of a dress, the jar of a step, feeling the pulse, are sufficient. Kussmaul correctly remarks that often a grosser irritation, such as pricking with a needle, causes no spasm, especially if the patient is prepared for it.

In traumatic cases it is stated that touching the wound will sometimes very readily excite an attack. A sort of aura is also described which sometimes arises in the wound before the attack.

The origin of these spasms has already been discussed, and but little remains to be added. The condition is one mainly of tonic spasms, which persist continuously while consciousness is retained, and must be looked upon as co-ordinated. This circumstance, as well as the order in which the muscles are attacked by the spasms, shows that they arise directly from the centres which control the spasms. The paroxysmal exacerbation of the spasms following insignificant irritations, as is seen in the later stages of the disease, must be looked upon as due to increased reflex irritability. Moreover, observation shows that the central inhibitory influence over reflex action is in its normal condition, for the attacks are more easily avoided if the patient is forewarned in regard to the sensitive impression.[1]

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[1] This predominating implication of the muscles of deglutition is not very frequent. *Rose* supposes that those cases are accompanied with spasms of deglutition in which the exciting injury is in the region supplied by the cerebral nerves. *Rose* designates these cases especially as cerebral tetanus, or tetanus hydrophobicus.

Compared with the spasms, motor paralysis is exceedingly rare in the course of tetanus. Some observations of Rose are interesting. He saw paralysis of the facial nerve in cases in which the injury giving rise to the attack was in the domain of the facial. This exception, according to the correct explanation of Rose, was doubtless dependent upon an ascending neuritis, which may all the more readily cause paralysis of the facial in view of the course which this nerve runs through the osseous canal. The circumstance that the paralysis was unilateral is greatly in favor of this view.

Other paralyses have not been noticed in the course of the disease; but, as terminations or sequelæ, not only have weakness and interference with muscular action from persistent stiffness been observed, but also paralysis.

Strabismus, which is sometimes noticed, is usually, according to Wunderlich, a precursor of death.

During the whole course of the disease the *intelligence* is characteristically clear and undisturbed; only exceptionally does delirium appear a short time before death. The higher senses are equally free from disturbance. Sleeplessness is another characteristic symptom, and is usually persistent; but if the patient does fall asleep, the spasms entirely cease, to recur when he awakes; the same is true during narcosis.

With the occurrence of the tonic spasms the *general health* is seriously disturbed; the difficult respiration, the contraction of the chest and œsophagus, the constrained and distorted position of the body, the impossibility of swallowing, with the presence of thirst and hunger, make the situation of the patient extremely distressing. All these tormenting symptoms increase during the paroxysms of spasm until they are unendurable, and the patient is thrown into a state of extreme anxiety and excitement. The same is true of the pain caused by the muscular spasms, which may often become very severe. A painful sense of pressure in the pit of the stomach is very constant; most patients complain especially in regard to this. Chalmer and others consider this pain as truly pathognomonic of the earlier stages of the disease.

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[1] A description of the changes in the muscles which occur during their activity cannot be given here; only the relations of the increased muscular activity to the metamorphosis of tissue are of unusual interest, and are therefore briefly mentioned.

The pains in the muscles have the same character as is found in other varieties of spasm, *e. g.*, cramp in the calf of the leg. Besides these cramp-pains, which are caused by the pressure of the contracted muscles upon the sensitive nerves, there is also hyperæsthesia. Tscharner says that pressure upon the contracted muscles excites severe pains; Demme also noticed increase of sensibility to pain.

On the other hand, Demme found very decided decrease of the acuteness of the sense of touch and of temperature. This observation stands almost alone in these respects, partly, perhaps, because the examination has not always been made; yet this change of the conduction of the sensitive nerves is certainly not frequent. Perverted sensations, or paræsthesiæ, are also reported in some few cases.

The stiffness of the muscles does not cause the above-mentioned sense of pain in all patients; sometimes it is felt only during the paroxysm. Indeed, single cases are reported in which during the severest attacks no pain is felt. G. Blanc mentions that in one patient the severest muscular contraction caused only a pleasant sensation of tickling.

The *skin* in most cases is almost constantly covered with profuse perspiration; this is greatly favored by frequent attacks of spasms. The explanation of this symptom is clearly this, that the increased muscular action increases the blood-pressure in the cutaneous vessels, and thereby the action of the sweat glands is increased. That this secretion of sweat exercises no marked influence over the course of the bodily temperature, though the vaporized moisture abstracts heat from the body, may be concluded from what is found in other varieties of diseases where sweating occurs with both high and low temperatures. The appearance of sudamina in connection with this is frequent, and is of no further significance.

A. Vogel noticed furuncles in one case, but referred their presence to large doses of bromide of potassium, which had been used.

During the persistence of cramp the *respiration* is only slightly changed in frequency; the patients breathe from twenty to twenty-four times per minute, or even less, but with greater effort and with a painful sensation of increased resistance.

The auxiliary muscles of respiration are affected by the spasm; the contracted abdominal muscles offer resistance to the diaphragm. Hence it happens that the secretion of the bronchi necessarily accumulates, and cannot be removed by coughing, since the preponderating action of the inspiratory muscles during the spasm seriously interferes with the falling-in of the thoracic walls from their weight and with the forced expiration necessary in coughing.

During the convulsions there is decided interference with the breathing; the patients become cyanotic and have the sensation of want of air, of danger of suffocation. The dyspnœa is caused in part by the spasmodic narrowing of the glottis, but especially by the spasm of the more powerful inspiratory muscles and the implication of the diaphragm. The thorax is spasmodically in the position of inspiration, the diaphragm is also drawn downward; thus the expiration is seriously restricted. In one case reported by Wunderlich the respiration was unusually slow, twelve to sixteen in the minute; during the paroxysms the respiratory action ceased, the abdomen projected forward, the intercostal muscles remained stiff. The return of respiratory action began with the diaphragm, which, though previously contracted, sank still lower. An opinion has already been expressed as to suffocation being the cause of death.

On account of the spasm of the glottis, tracheotomy has been proposed and performed, but always with unfavorable effect, for the pulmonary symptoms (bronchitis and accumulation of secretions) increase to a dangerous degree afterwards.[1]

The *pulse* during tetanus may be entirely normal, or below the normal number,[2] and between the paroxysms it is, as a rule, normal or only slightly accelerated. But the frequency varies, depending upon the attack of spasm, since with every shock the pulse rises ten or twelve beats, and becomes lower when this relaxes. Exceptions are sometimes found; thus Hennen, Macgrigor and Morrison saw the pulse unaffected during the severest attacks. But during the paroxysm the frequency of the pulse may be much increased, it may even reach 180. This enormous frequency is seen most often in the last stage, and at that period the symptom does not depend upon the spasms, which only cause an additional increase in the otherwise high pulse; thus Griesinger in one case noted a pulse of 170, while during sleep it was 140. This excessive frequency is indeed the result of many factors; it only remains to inquire which is the more important. The simultaneous presence of a high temperature certainly has an influence. Yet probably an abnormal action of the cardiac nerves is the most important factor, though at present it is not possible to decide whether there is diminution of the tone of the pneumogastric nerve or irritation of the sympathetic. In favor of the latter supposition is the quality of the pulse, which is found to be small and irregular, pointing to a vaso-motor irritation.

According to Liston the vessels may be contracted to such a degree that even amputation may be performed without the loss of a drop of blood. The contraction of the vessels reacts upon the heart as an accelerating cause through the increase of resistance. The resistance is also considerably increased during the attacks of spasms by the muscular contraction, so that the increase and decrease in the frequence of the pulse is dependent upon the accession and cessation of the spasms.

The *heart* may suddenly cease acting during the attack; this can be determined by experiments upon animals, and agrees with observations upon patients. This does not denote the existence of tetanus of the heart, as is evident *à priori*, and is still further proved by the cessation which occurs during diastole. Probably the cause is to be found in the sudden increase of resistance, especially as there is also exhaustion and cardiac debility; or there may be irritation of the pneumogastric.

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[1] Med. Times and Gaz. London, 1854. p. 462.

[2] See *J. W. Ogle*, Transact. of the Clinical Soc. 1872. Vol. V.

Increase of temperature in tetanus has been recognized for a long time; it was noticed by de Haën, then by Bright and Prévost. Nevertheless, opinions have been very much divided as to whether tetanus is a febrile process or not; thus Hillary, O. Beirne and others asserted that tetanus ran its course without fever. Lately this fact has been made the subject of so numerous investigations, under the stimulus of Wunderlich, that the result may be summarized thus: Tetanus causes no increase of temperature or only a slight increase, and this, according to J. Ogle, occurs especially in the evening.[1] Of course, there may be fever if the wound or some other complication causes it. On the other hand, in many cases of this disease it is found that the temperature rises to an excessive height, and this exacerbation occurs generally a short time before death—in one case of Wunderlich it was 44.75° C. (112.73° F.) in the axilla; it has also been found that after death the temperature continues to rise for a considerable time. This elevation of temperature shortly before and after death has been frequently noticed since Wunderlich, but it may also be wanting in fatal cases of clearly marked tetanus (J. Ogle, E. Rose).

An understanding of these relations has been much favored by experiments upon animals. The investigations of Leyden, then of Billroth and Fick, agree perfectly in their results: In tetanus caused by electrical irritation of the spinal cord the temperature rises enormously, and also continues elevated after death (Billroth and Fick). In each attack the temperature sinks somewhat during the first moment, then it rises continuously, and, indeed, this rising lasts some time longer than the muscular contraction. Before the contractions the temperature is lower in the muscles than in the rectum, but with the spasms this relation is reversed.

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[1] Transact. of the Clinic. Soc. V. 1872.

The same occurs in tetanus from strychnia poisoning.[1]

These high temperatures, as they are seen in many cases during the final stage of tetanus, have been referred by many to the muscular spasm, therefore to *increased production of heat.* When a muscle falls into a state of contraction it not only becomes demonstrably warmer, but there is in truth more heat produced in the acting muscle. The increase of temperature during muscular action is found not only in the muscles themselves which are in action, but also in the whole body; but in the body collectively the increase is very slight (according to Davy, 0.70 C. [1¼° F.]).

The increase of temperature in muscles in a state of activity was proved by Becquerel and Breschet, then with better methods by Helmholtz. Solger has reported the increase of temperature in excised muscles, and also the diminution at the beginning and the continued increase after the cessation of contraction, and has designated these phenomena as negative temperature variation and as supplemental increase;[2] but, according to Heidenhain, the temperature increases immediately with the beginning of the tetanus, and the negative variation is an error of observation.[3] Ziemssen has shown the increase of temperature also in the layer of skin which lies above the contracting muscle.[4]

These facts do not, however, conclusively prove that more heat is really produced during the activity of muscles; that the rise in temperature is really caused by increased production of heat. Billroth and Fick, indeed, also found that during an attack of tetanus the temperature rose much more rapidly in the muscle than in the rectum, while during rest the temperature of the group of muscles examined was lower than that of the rectum. These observations are enough to render it probable that there is an actual increase in the production of heat during muscular action, and this view is more firmly established if the products of tissue change during rest and during action are compared. Voit has shown that muscular action is not accompanied with any marked increase in the decomposition of albumen in the entire body, while, according to Pettenkofer and Voit, the excretion of carbonic acid is very much increased during muscular action. Thus, during action more heat is produced, besides the mechanical power, and, indeed, the excess may equal the entire heat-producing power of the non-nitrogenous substances which are more decomposed in consequence of the muscular action, while probably the production of mechanical action belongs only to the albuminoid substances, and a transformation of heat into mechanical power within the body is improbable.

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[1] *Guentz*, Beobachtungen über die Temperatur beim Tetanus; and *A. Muron*, De la cause de l'élévat. Gaz. méd. 1872.

[2] Stud. aus dem physiol. Instit. zu Breslau. 2. Heft.

[3] Mech. Leistung, Wärmeentwickelung u. s. w. bei der Muskelthätigkeit.

[4] D. Elektricität in d. Medic.

The explanation of the extraordinary temperature in tetanus, on the ground of muscular action, seems to obtain still further support from the circumstance that in it the muscular action consists chiefly in tonic spasm. A muscle in a state of tonic contraction, according to J. Béclard, produces more heat than when employed in ordinary work, and, according to Ch. Bouchard and J. M. Charcot, spasmodic actions may be divided into two groups with reference to variations in temperature, viz.: one group with static contractions, which cause a greater or less increase in temperature; the other, with dynamic contractions, which have no influence upon the temperature. Such a distinction is, however, by no means always correct. But if the above-mentioned influence upon the temperature is ascribed to tonic spasm, on the ground that such spasm accomplishes no external work, this is surely a mistake.[1]

But in settling the question of the increased production of heat during muscular activity we by no means settle the matter of the abnormal elevation of temperature during tetanus; indeed. the most cogent reasons exist against accepting the one as an explanation of the other. We know how slight an increase of temperature occurs from severe muscular action. Furthermore, clinical observation shows that in tetanus, notwithstanding the most severe spasm, the temperature may be, and generally is, normal until it rises a short time before death, often simultaneously with a cessation of the contractions.

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[1] The error, indeed, is evident from the simple consideration that that amount of heat which corresponds to the mechanical work accomplished by a man is so slight that an increase of the temperature of the body cannot be explained thereby, especially as the specific temperature of the body (the muscle) is very high.

The possibility of a regulating power which maintains the temperature of the body at about a certain standard, notwithstanding varying degrees of heat-production, exists in tetanus just as well as in a normal condition, as is proved by the fact of the temperature remaining normal in spite of the muscular cramps. It is certainly more correct to refer the increase of temperature towards the end of life to a disturbance of this regulating power than to an increased production of heat beyond the limit which can be compensated for.

The extraordinary high temperature occurring just before death does not belong exclusively to tetanus. The same condition is found likewise in injuries of the cervical vertebræ, the most instructive example of which was observed by Brodie (dislocation of the fifth and sixth cervical vertebræ, with a temperature of 43.90 C. [111.02° F.] without muscular spasm). Very nearly the same is seen in other cerebral and spinal affections, as is shown in cases reported by Wunderlich.[1]

The question of the regulation of the temperature of the body is as yet by no means settled. It is a fact that complete division of the spinal cord in the neck causes the temperature rapidly to sink, unless the rapid loss of heat is prevented by external means. A communication is thereby interrupted in the cervical portion of the cord which, normally proceeding from the central system, prevents the rapid loss of heat. If, however, the communication is not interfered with, and the centre is irritated, the temperature rises. The further details of the question are unimportant for our purpose; yet, for the present, we may regard the vaso-motor centres as among the organs governing the regulation of heat, without danger of falling into any serious error.

Hence we are led to the conclusion that the excessive increase of the temperature in tetanus arises from an irritation of a centre of innervation which normally is concerned in effecting the escape of the variable amounts of heat produced within the body. At the same time the production of heat is increased by the exaggerated muscular action.

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[1] Archiv der Heilkunde. 1864. Bd. V. See also *Guentz*, Allg. Zeitschr. für Psych. 1868.

A post-mortem increase of temperature has been noticed since de Haën after death from various causes; only in tetanus alone it is common for the post-mortem increase to be very great, as Wunderlich first stated. The reason for this elevation of temperature after the cessation of the circulation, according to Huppert, and also according to Fick and Dybkowsky, is to be found in the setting free of heat during the stiffening of the muscles, on the coagulation of the myosin. At the same time, with the cessation of the circulation, the circumstances governing the giving off of heat change, the exchange from one layer of the body to another is accomplished by conduction without the intervention of the circulation.

Processes of Decomposition.

Condition of the urine.—The amount of urine passed in twenty-four hours in tetanus is usually below the average in health, even in persons fasting; on cooling, there is generally a deposit of the urates previously held in a saturated solution by the elevated temperature. The reaction is strongly acid, corresponding to the concentration (only exceptionally alkaline by decomposition), and the specific gravity is proportionately high. The urine is rich in brown coloring matter.

Exceptional cases occur in which large quantities of urine containing but little coloring matter are evacuated, but these depend probably upon complicating circumstances. Tetanus is then distinguished, by the amount of urine excreted, from some other forms of spasms with a large amount of urine (urina spastica). Textural diseases of the kidneys can hardly play any part here, but a number of other conditions coexist with this diminution of water in the urinary secretion, such as profuse perspiration, difficulty in drinking, diminished pressure in the aorta. Experimental investigation has not yet settled most of the questions concerning the influence of the renal nerves upon the action of these organs, while the varieties of spasm plainly point to such an influence. The experiments of Cl. Bernard, Eckhard, and others, are as yet not sufficient for pathology.

Albuminuria occurs, but not habitually; the proximate causes of this may, indeed, vary; it may result from congestion, especially in the later stages, or from complication with renal diseases, which occur independently of the tetanus, as must be supposed in the case reported by Kussmaul.[1] As yet too little is positively known in regard to the influence of innervation to take it into account as a factor. Albuminuria is not a frequent occurrence in tetanus; very many negative facts are found in medical literature, and in five cases wherein I examined the urine I found no albumen.

An observation of Griesinger is deserving of mention. In a case of traumatic tetanus he found in the urine, which had a dense deposit, numerous pale casts, without albumen. At the autopsy there was found only desquamation of the pyramidal portion.[2]

The presence of sugar in the urine in tetanus is also of interest. This was first noticed by Demme. A. Vogel has also described a case of rheumatic tetanus in which there was sugar in the urine. Vogel refers the phenomena to irritation of the spinal cord.[3] This is, however, very rare.

The urine in tetanus generally possesses, to a very great degree, the peculiarity of reducing the oxide of copper, but without depositing oxydul. The fluid changes color by boiling, and takes a clear dark-yellow color. The amount of this reducing substance is, of course, only dependent upon the concentration.

The excretion of nitrogen in the urine in tetanus was first determined by Huppert in a case of rheumatic tetanus. The variations in the daily amount of urea were so great (45.2 and 12.2 grms.) that they could only have been dependent upon the absorption of varying amounts of nourishment. The demand for nourishment, according to his statements, was very active, but the appropriation of the nourishment was much interfered with.[4] On the contrary, Senator, after examining two cases, came to the conclusion that there was no increase in the excretion of nitrogen as compared with that in a person who was fasting; that the course of the decomposition of albuminoid matter was independent of the attacks of spasm, and that it was not even clear that the increased temperature exercised any influence thereon.[1] I arrived at the same result in two cases where I carried out experiments to determine the decomposition of albumen.

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[1] Berl. klin. Wochenschrift. 1871.

[2] Arch. der Heilk. 1862.

[3] Deutsch. Arch. für klin. Medic. Bd. X.

[4] Arch. der Heilkunde. 1862. p. 177.

Hence also in the pathology of tetanus the proportion established by Voit holds good, that the activity of the muscles takes place independently of the decomposition of albumen.

Among the components of the urine containing nitrogen *creatinine* is of interest on account of the relations to muscular action ascribed to it by earlier observers. Senator, in his cases, found but a very small quantity of creatinine in the urine, agreeing with the facts established by Nawrocki for individual muscles, and more extensively for the whole body by Voit. Creatinine or creatine, therefore, stands in no relation to the activity of the muscles; hence in general tetanus there is no increase in its amount.

Senator even found the amount of creatinine diminished in comparison to that of urea; this would support the view that by muscular action a portion of the creatine and creatinine is transformed into other compounds, which is quite possible, according to the data given by Voit.

No direct estimate has been made of the amount of *carbonic acid* produced through the decomposition of the non-nitrogenous substances. Yet from other well-known facts it may be inferred with great certainty that it is increased proportionately with the muscular action.

In regard to the digestive organs it may be mentioned that the tongue is generally dry and coated, the saliva is tenacious and ropy.

The thirst is very great and distressing on account of the loss of water through the skin and the difficulty in taking liquids. The desire for food is often not at all diminished, there is the most acute hunger, and food is well borne when the stiffness of the jaws does not prevent its being taken. On account of the above difficulty, therefore, the patient may actually suffer severely from hunger. This fact shows that the gastro-intestinal digestion is at least not always deranged.

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[1] Virchow's Archiv. Bd. XLVIII. 1869.

Constipation is a common symptom in tetanus; the fæces also have an intensely strong odor and an unusual appearance ("like sloughs"), depending upon the length of time they have remained in the intestines. The constipation is probably dependent upon the rigidity of the abdominal muscles and anomalous peristaltic action, but only in a slight degree upon the spasms of the sphincters. The anomalous peristaltic action may be found in the failure of regular propulsive motion; evidently cessation of motion as well as persistent spasm may check the propulsion of the contents of the bowels, and it is quite possible that in tetanus the muscles of the intestines may be in a state of spasm.

Micturition is not seldom affected, and the urine cannot be voided voluntarily; involuntary micturition during the paroxysm or as a symptom of paralysis during the later stages is more rare. This is more common among children. According to E. Rose the retention of urine depends upon the deficient action of the abdominal walls, and this is certainly a cause, but not the only one. There is at the same time also spasm of the sphincter vesicæ, or, rather, a loss of ability to relax that muscle. The abdominal muscles are indeed in a state of rigidity, but they are not withdrawn from the control of the will. The retention of urine is also by no means constant, and will frequently cease spontaneously if the patients are put into a warm bath for a while.

Terminations and Prognosis.

The symptoms described may be present with more or less severity in individual cases, they may also in part be wanting; and it is the variations in this respect which give to each case its individual character and mark the intensity of the disease. Thus, there are extremely light cases in which, besides trismus, there is at most only some stiffness of the neck; such cases occur especially from the effects of a chill. In adults their termination is favorable, yet it cannot be positively foretold whether there will be a further development of the symptoms; but in children even trismus is very dangerous.

So soon as the symptoms are developed to their full extent, the danger to life is extremely great, and among the newly born the fatal termination of tetanus is so constant that the occasional cases of recovery have been looked upon as being probably errors in diagnosis; still, individual cases of recovery are certainly seen. Among the different symptoms there are also certain whose presence is peculiarly significant of danger.

Most physicians consider the rheumatic variety as less dangerous than the traumatic; by others, however, this, too, is denied.[1] It is difficult to form a definitive opinion in regard to the rate of mortality in tetanus. In the first place it may be questioned whether in certain collections of statistics the milder and insignificant cases have been included; then the collection of the cases separately published is open to the objection that the favorable cases are more frequently published than the unfavorable, as the object generally is to call attention to the results of therapeutic measures. Thus, the tables given by Friedrich and Curling do not harmonize with the army reports. The truth will be found to lie midway between the two, as the latter show too high a rate of mortality on account of the unfavorable circumstances which are connected with the care of the wounded in war.

In the tables given by Curling, of 128 patients 58 are noted as recoveries; by Friedrich, of 252 cases 128 proved fatal. On the other hand, Macgrigor, in his report of the English and Spanish war, declares that among some hundreds of patients there were only few cases of recovery. According to H. Demme, of 86 cases of tetanus in the Austrian and Sardinian-French war 80 were fatal.

According to an old surgical rule, tetanus is the more likely to end favorably the longer the period of time between the causative injury and the occurrence of the tetanic symptoms. Statistics justify this statement; for, according to Poland's collection in Guy's Hospital, 96.7 per cent., and in the Glasgow Hospital, 96.6 per cent. of the cases of tetanus proved fatal when the symptoms appeared within ten days after the injury, while the entire mortality is 84.2 and 87.5 per cent. (E. Rose).

Different authors ascribe peculiar prognostic value to single symptoms. Thus, Laurie says, those cases are especially serious where during the first days of the disease every attempt to swallow causes an attack of suffocation. Wunderlich sees the first signs of the fatal catastrophe in the occurrence of a slight strabismus; the danger of a very frequent pulse is especially insisted upon by Parry. The prognostic value of these and similar separate signs is, however, always rather limited. Yet almost all physicians agree in this, that where the paroxysms are severe and of frequent occurrence, there is great danger to life.

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[1] The Dublin Journal of Med. Science. 1872. III. p. 185.

A quickly fatal termination is the rule in cases where the rigidity rapidly increasing becomes very great—in the so-called acute cases, according to E. Rose. Cases are recorded where death occurred in the short space of a few hours; thus, Whytt saw a young girl die after ten hours. Especially in the newly-born death generally follows in a very short time. Most of the fatal cases occur within the first ten days from the beginning of the disease. Among Friedrich's 128 fatal cases 83 died within the first four days, and according to the statistics of Poland, covering 262 cases, 63 per cent. died during the first five days, and 88 per cent. during the first ten days (E. Rose).

Hence we deduce an important prognostic rule, namely, that after the first days the probability of recovery rapidly increases. This has been long known, and found expression in the Hippocratic aphorism, according to which tetanus ends in recovery if the fourth day of the disease can be survived. This rule is, of course, not true within such narrow limits, as death may occur, exceptionally, even after the course of weeks. These exceptions depend upon the fact, either that the paroxysms of spasms are renewed after they had once ceased, or death takes place from exhaustion and complications. The paroxysms of spasms exert a special influence upon the extreme mortality during the first stage of the disease; their long continuance renders life impossible; and hence, if their severity does not quickly diminish, they prove fatal. In fact, death frequently occurs during an attack. In other cases the spasms cease, and death follows during a mild delirium, generally with greatly increased frequency of the pulse and high temperature.

In cases of recovery the convulsive attacks diminish, becoming lighter and less frequent, and after a time entirely cease. The rigidity still continues for a while; it disappears very gradually, and not in any particular order. Hence, it is generally many weeks before complete recovery; indeed, a certain irregularity in muscular action, the sense of stiffness, may persist quite a long time. Possibly ruptures and hemorrhages have an influence in this direction. The return of sleep is also a favorable indication.

In certain cases of tetanus permanent consequences, paralyses, have been reported. This is, however, very rare if, indeed, the accuracy of these observations may not be doubted.

Diagnosis.

In well-marked cases the features of tetanus are so clear that there is no difficulty in the diagnosis. Yet, at the beginning of the disease the obscurer symptoms may be easily overlooked, or their importance underestimated; in cases of disease among nurslings, especially, the disturbance in nursing should lead to local examination. Then, too, it is possible to confound this disease with some others, both in its beginning and during a more advanced stage; finally, there are cases which, according as views differ, will be called tetanus by some and a different disease by others.

In by far the majority of cases trismus is the first symptom of tetanus; rarely is it the only one. But as the stiffness of the jaws may occur from various circumstances, it is especially necessary to examine whether it may not depend upon some local cause, as angina, parotitis, disease of the teeth or of the articulations, etc., and this is not a difficult examination.

The diagnosis of those cases of tonic bilateral spasm in the domain of the trigeminus, which depend upon other causes, and are not to be regarded as tetanus—the so-called *masticatory facial spasm* (see this Cyclopædia, Vol. XI. p. 301)—must depend upon a consideration of the etiology and the accompanying symptoms. Furthermore, the statement of E. Rose is of value here, according to which, even in the light forms of tetanus, the cervical muscles are slightly affected, so that simple trismus must be considered of rare occurrence when the conditions are present which may give rise to tetanus.

It has been already stated that tetanic spasms may occur in connection with a great number of morbid processes, and that the succession and order of the spasms especially determines the diagnosis. Thus it is easy to recognize general convulsions as in uræmia, likewise other forms of spasm, as epilepsy, paralysis agitans with muscular stiffness, etc. Hysterical spasms, too, can easily be distinguished from tetanus, though the resemblance is so great that a hysterical tetanus has been described; but an interval of uncertain length, which is entirely free from spasm, follows the paroxysms in hysterical disease, and then the hysteria can also be recognized by other symptoms. Diseases of the spinal cord, accompanied with symptoms of motor irritation, especially myelitis, are likewise not difficult to distinguish from tetanus by the order of succession of the spasms and the paralytic symptoms which appear at the same time.

In certain stages tetanus may be confounded with cerebrospinal meningitis, even with tubercular basilar meningitis, as both give rise to stiffness of the neck. But in the two latter there is rarely trismus, and the accompanying symptoms of both processes would prevent any prolonged mistake.

That form of spasm known by the name of tetany can be sufficiently distinguished from tetanus by the very different succession of the spasms, the perfect intermissions, also by Trousseau's symptom and the result of electrical examination (see this Cyclopædia, Vol. XI.).

It is of interest and important to recognize during life the difference between essential tetanus and cases of toxic origin, especially strychnia poisoning. (For a more accurate account of this, see this Cyclopædia, Vol. XVII., also the proof of the presence of poison in cases of poisoning.)[1] The principal diagnostic points may be summed up as follows: Essential tetanus begins with trismus, and gradually descends to lower muscles; the increase of reflex irritability is at first not very marked; on the other hand there is no intermission of the tonic spasms. In strychnia poisoning the general reflex spasms appear immediately in their full intensity, but there are perfect intermissions, and they soon end in death or recovery. Besides, in the tetanus caused by strychnia, the spasms chiefly affect the extremities, the hands, but in tetanus it is not so. In strychnia poisoning there are gastric symptoms, and consciousness is lost, symptoms which do not occur in tetanus. These diagnostic differences are not so clearly marked as the above statements would lead one to suppose, and they are well defined only in cases of acute poisoning when compared with the lighter cases of tetanus; in chronic poisoning the phenomena are different.

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[1] Compare also *L. Hermann*, Lehrb. der Toxikologie. 1874.

It is scarcely necessary to mention that tetanus must not be confounded with hydrophobia, as formerly happened; the latter resembles tetanus only in the reflex spasm of the muscles of deglutition, but otherwise the two diseases have little in common. (See this Cyclopædia, Vol. III.)

Treatment.

The history of the treatment of tetanus is very instructive, showing how little prospect there is of establishing any therapeutical proposition by purely empirical means. There is scarcely a remedy, especially if it has any influence upon the nervous system, which has not been used in the treatment of tetanus, and been praised for its success. On the other hand, we see again also that so-called empiricism can never be without theoretical ideas, and is always founded upon them.[1] Thus the notion that tetanus was of an inflammatory nature, led, as a natural consequence, to the use of antiphlogistic remedies: bleeding and mercury. Mercury was highly recommended by Donald Monro, and was subsequently often used even to salivation; but Larrey, J. M'Gregor, Mosely and others, declared that its use was without good effect and injurious, and cases have been reported in which, indeed, the outbreak of tetanus was connected with the fact of the salivation. It is scarcely necessary to say that mercurial stomatitis occurring with the tetanus is not a matter of ndifference. General blood-letting, which formerly retained its reputation in the treatment of tetanus, notwithstanding many contra-indications, has in a great measure lost its reputation, owing to the lack of inflammatory changes found in the body. Tetanus is no longer treated by blood-letting, unless it be in Italy. The spasms may, indeed, temporarily cease after bleeding to syncope, but it is scarcely necessary to refer to the irritating effect of cerebral anæmia upon the motor centres. Besides, there are numerous other reasons which forbid extensive bleeding in the treatment of tetanus. Individual cases of recovery are not sufficient to offset these contra-indications.

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[1] See Med. therap. Wörterbuch von *Ch. Siegert.* 1856.

No attention need be paid to this nor to an entire series of similar procedures in treatment. I believe that the symptoms of tetanus present very decided indications, since there cannot be any question of a truly specific line of treatment.

As tetanus is of traumatic origin in the vast majority of cases, and as we cannot avoid the conclusion that improper treatment of the wound and unfavorable hygienic conditions have great influence upon the occurrence of the disease, it is indispensable to use all possible care in the treatment and care of the wounded, according to the well-known principles of surgery, and especially is the influence of sudden and severe changes of temperature to be avoided.

If a case of tetanus is dependent upon the lesion of a peripheral nerve which can be reached, it seems necessary to cut short the peripheral irritation according to the best of our ability. This proposition has been long recognized, and the solution of the problem has been sought in various ways. Amputation and excision must appear the most certain means to eradicate the evil by the roots ; these procedures have often been put in practice, and were especially praised by Larrey, and even at the present time the discussion in regard to them has not ceased. Clearly this question only relates to secondary operations, when the symptoms of tetanus are coming on or are already present; that is, to operations which are performed on account of the tetanus, and for no other reason. But the most satisfactory reasons exist in opposition to amputation ; the wound left thereby itself again presents a seat of irritation for the ends of numerous nerves, and these may under some circumstances give rise to tetanus even after primary amputations. Then the discovery of ascending neuritis, with all its consequences, would contraindicate amputation, as the object sought by the operation would not be gained. Among the opponents of amputation we find the most celebrated surgeons, such as A. Cooper, Dupuytren, and others. The indication present should be fulfilled by milder measures, by division of the nerves, which was first done by G. Hicks. The most striking results of this method were obtained by Murray, and many examples of its favorable influence have since been reported. The only objection to the general practice of immediate section of the nerve may be found in the fact that it is frequently necessary to follow it by more extensive surgical interference, and that, if there is ascending neuritis, the source of the disease is not reached.

Nevertheless, surgical treatment opens a wide and influential field: the careful treatment of the wound itself, removal of foreign bodies, dilatation of wounds where the secretions are retained, in some cases resection of the nerves, are of the greatest and most pressing importance. It is especially necessary that every manipulation of the wound should be under the influence of an anæsthetic. Again, the nerve-trunk if already inflamed should be handled as little as possible, and we should hardly attempt to get along without the local use of morphia.

A further indication is to diminish or remove the central irritation, and to hold in check the excessive irritability already present. The first of these purposes cannot be attained in any better way than by the use of narcotics and hypnotics. Among the different agents of this class opium and morphia are the ones almost universally used, and, indeed, by many physicians in enormous and even incredible doses. Both agents have certain properties which render them objectionable for this purpose, *i. e.*, their primary stimulating effect, which may manifest itself by exciting spasms, though this is rare in man. In fact, they are practically and properly being more and more supplanted by *chloral hydrate;* this is superior to every other means, even the inhalation of chloroform, for the accomplishment of the purpose in hand. Hashish or cannabis Indica, which has been recommended by some physicians, is too uncertain as a hypnotic, and its action is not sufficiently well known to justify its use in this connection. *Tobacco*, which has been highly praised in the treatment of tetanus by many physicians (especially Curling), exercises a favorable influence upon the spasms by its powerful paralyzing action upon the cerebrum and spinal cord. But it also possesses serious disadvantages, especially by its primary stimulating action, and the extreme depression which follows its use. Hence, the desired object of diminishing the irritability can be better attained by the use of *bromide of potassium*. *Calabar bean* or *physostigmin* is also to be recommended for this purpose, as it at the same time also diminishes the irritability of the motor nerves.

As the tetanic spasm may evidently also be affected if only the irritability of the *motor nerves* and of their termination in the muscles be diminished, various remedies have been used in tetanus which act in this manner. Among these are included *prussic acid*, *belladonna*, and especially *curare*. In controlling the spasm we do not, of course, consider the tetanus as cured, but only that one symptom is removed, the importance of which has been sufficiently discussed. Cases enough are known where the fatal termination has followed, notwithstanding the interruption of the spasms. This fact, of course, will govern the choice of remedies so that other injurious consequences may not follow their action, especially in regard to the heart. Considering these circumstances, the control of the spasm will be attained most satisfactorily by the use of hydrate of chloral and bromide of potassium.

The cold bath and cold douche must be looked upon as antiquated and heroic means, whose efficacy, in their time, was regarded by Wright and Currie, and especially by Brodie, as beyond all doubt. Certainly, cases have been known in which a severe attack and consequent death have occurred when the patient was put into the water, but there have also been cases of rapid improvement. That this procedure is, in truth, dangerous, is evident from the fact that the influence of cold is a powerful irritant of the sensory nervous system. But the possible efficacy of this method must be sought in this same powerful sensory irritation, as the severe excitation must produce subsequent exhaustion; hence an influence must be exerted upon the distribution of the blood and upon the nerves of the vessels. However, the possible danger attending their use is an argument against having recourse to baths, but they are a very valuable agent, and I ought to point out a special indication for their use, viz., that stage of the disease accompanied with excessive elevation of temperature, often combined with cessation of the spasm, delirium, etc. Under these circumstances a bath, especially one with gradual reduction of the temperature, meets the double indication of lowering the temperature and exerting a reflex influence upon the brain, etc. *The warm full bath*, especially recommended by French practitioners in the rheumatic form, can have scarcely any other object than to equalize the irritability of the sensory nerves, change the distribution of the blood, and prevent variations in temperature. The fact is, that patients find the warm water very pleasant, and on this ground its use is certainly justified, though the experience of different physicians varies in regard to its influence upon the spasms and the course of the disease. *The vapor bath*, likewise employed, offers the same advantages only in less degree, but the objections to it are greater. (Stuetz's alkaline bath is antiquated.)

To prevent the spasms, care must be taken to put the patient under such conditions as to avoid all irritations, as noises, jarring, changes from light to dark, and changes in temperature. The patient should be forewarned of every unavoidable touch.

A very important task is to support the strength; by most observers the greatest emphasis is laid upon this, to place at the disposal of the organism as much power as possible by means of food and stimulants. Feeding is rendered very difficult by the closure of the jaws, the introduction of solid food is often impossible, and even the administration and swallowing of fluids may be prevented. Hence, the proposal is quite rational in such cases, during anæsthesia, to introduce food through the stomach-tube, perhaps through the nostril. Besides, the difficulty caused by the locked jaw may be met by the use of nutritive enemata.

Even to the present day English physicians emphasize the importance of caring for the free evacuation of the bowels. There are certainly some arguments in favor of this, for an evacuation of the bowels rarely occurs spontaneously, and the accumulation of fæces is accompanied with a series of consequences which may most disastrously influence the result in tetanus. Probably this object can be attained without danger by the exhibition of powerful cathartics according to the English custom, yet an energetic irritation of the intestines in this way does not harmonize with the other indications, and had better be avoided.

The treatment hitherto mentioned fulfils the indication of relieving the patient's suffering and overcoming the sleeplessness. The field of therapeutic effort is not in all cases exhausted thereby; individual symptoms may still require special treatment, as rapid pulse, retention of urine, impending suffocation, immoderate sweating, etc.

The use of electricity in the treatment of tetanus is still to be mentioned. As yet few trials have been made with this agent; these are in part negative, in part positive in their results; the question is still open.

The treatment of *tetanus neonatorum* must be conducted according to the same principles, of course with the changes rendered necessary by the difference in age; the very few cases of recovery have been ascribed chiefly to the opium treatment.

CATALEPSY.

EULENBURG.

CATALEPSY.

Wedel, De affect. soporosa et catalepsi. Jena, 1708.—*Tissot*, Œuvres. t. XIII. c. XXI.—*J. Frank*, Præcepta II. vol. I. sect. I. p. 478.—*Georget* and *Calmeil*, Dict. de méd., Art. Catalepsie.—*Leubuscher*, Ueber Abulie. Damerow's Zeitschr. 1847.—*Blasius*, Ueber Stabilitätsneurosen. Archiv d. phys. Heilk. 1851. S. 234.—*Skoda*, Zeitschr. der k. k. Ges. Wiener Aerzte. Nov., 1852.—*L. Meyer*, Charité-Annalen. 1854. H. 3.—*Puel*, Mém. d. l'Acad. de Méd. 1856.—*Buchanan*, Glasgow med. journal. 1857 and 1858.—*Schwartz*, Rigaer Beiträge z. Heilk. 1857. Bd. IV. S. 118.—*H. Meissner*, Archiv d. Heilk. 1860. S. 572.—*Mosler*, Virchow's Archiv XX. 1861. S. 15.—*Jones*, British med. journ. 6. June, 1863. —*Macedo*, Siglo medico. June, 1864.—*Lasègue*, Arch. gén. de Méd. 1864. I. 1865. II. p. 385.—*Hartmann*, Würtemb. med. Correspondenzbl. 1866. No. 35.—*Smoler*, Memorabilien, No. 7. 1866.—*M. Rosenthal*, Wien. med. Presse. 1867. No. 5.—*Haase*, Archiv d. Heilk. 1868. H. 5. p. 492.—*Stiller*, Wiener med. Wochenschr. 1868. Nos. 103 and 104.—*Hasse*, Krankheiten des Nervensystems. 2d edition. Erlangen, 1869.—*Benedikt*, Elektrotherapie. 1869.—*M. Rosenthal*, Handbuch der Diagnostik und Therapie der Nervenkrankheiten. Erlangen, 1870.—*Handfield Jones*, Studies on functional nervous diseases. London, 1870.—*Holm*, Nord. med. Arkiv. III. Nos. 14, 19. 1871.—*Jamieson*, Edinb. med. journ. July, 1871. p. 29.—*Corlieu*, Union méd. 1871. No. 69.—*Walcher*, ibid. No. 74.—*Glas*, Upsala Läkareforen. Forh. VII. p. 17.—*Wilhelm*, Wiener med. Presse. 1872. No. 30.

By catalepsy is understood a diseased condition of rare occurrence, which is paroxysmal in its manifestation. Its pathognomonic symptom consists in this, that *the muscles remain in a state of contraction into which they enter at the beginning of the attack, so that certain positions of equilibrium are maintained by the limbs without spontaneous change, while passive changes of posture, sometimes of very singular nature, can be easily made* (so-called flexibilitas cerea). As a rule, the attacks are accompanied with diminution or loss of sensation and consciousness. The affection occurs, on the one hand, as one of the many manifestations of hysteria, on the other hand, as a symptom of chronic cerebral disease (softening, tubercular meningitis) and poisoning, but also as an independent uncomplicated affection.

Synonymous terms are: Morbus attonitus (Celsus); synochus; eclipsis; hysteria cataleptica; trance. (By the latter name some authors understand especially attacks, occurring in persons of an hysterical disposition, which are midway between true catalepsy and somnambulism.)

Etiology.

The causes of catalepsy in hysterical patients are the same as those of the hysteria; this may also be said in regard to the cataleptic phenomena when occurring as symptoms of chronic softening, tubercular meningitis, etc. The etiology of uncomplicated, idiopathic catalepsy is almost entirely unknown. We venture to assume that catalepsy belongs to the large group of diseased conditions designated by Griesinger constitutional neuropathies, whereby its near connection with other neuroses of this group (hysteria, insanity, epilepsy, chorea, etc.) is indicated, and also that a predisposition dependent upon congenital preformation of certain portions of the central nervous system generally precedes the appearance of the cataleptic attack. This supposition is yet further supported by the fact that we not rarely observe catalepsy in families in which certain members are disposed to other nervous disturbances of this group, especially to chorea and epilepsy; or that the person afflicted with catalepsy is also subject to other, especially to convulsive neuroses of the most varied nature; or that primarily epileptic attacks preceded the cataleptic. Likewise the circumstance is of importance that catalepsy, like other neuroses depending upon constitutional predisposition (as chorea, epilepsy, hemicrania), may appear at a very early age. Catalepsy has been noticed at five years of age.—As to the influence of age, the disease is most frequent in youth, especially at the time of puberty; in advanced age catalepsy occurs only exceptionally. There does not seem to be any marked difference in predisposition between the two sexes. The supposed preponderance of females depends upon the influence of hysteria, which, indeed, furnishes a large contingent to the cataleptic condition in its widest sense. Besides, chlorosis also may possibly exercise a causative influence among women.

Of the more accidental influences which may give rise to catalepsy, first to be mentioned are strong emotions, the influence of which is almost undisputed. For example, Jones saw a case of catalepsy in a man sixty years old, caused by his horror at the sudden death of his wife; Rosenthal saw a case in a boy twelve years old, which came on after great excitement. Further, over-excitement in certain spheres of mental action undoubtedly has a certain value as an etiological factor; especially is this true of fanatical devotion to certain religious ideas, mystic subtilties, and speculations, though it must not be denied that, on the one hand, such a mental bias itself in certain cases is often to be looked upon as a manifestation of a diseased, unnatural nervous action. On the other hand, also, the etiological connection appears more clearly with the nearly allied condition of ecstasy and somnambulism than with catalepsy proper.—In certain cases malarial infection is mentioned as the cause of typical attacks of catalepsy; also dreams and atmospheric influences (probably, however, only with previous predisposition) may excite catalepsy. Thus, Schwartz noticed in a boy seven years old, in consequence of rough maltreatment, first, a choreic condition, which later passed into catalepsy. Jamieson saw a cataleptic attack in a girl eighteen years old, after a severe blow on the right side of the back—yet the same girl, two years earlier, had a similar attack after a fright. Hartmann observed catalepsy after evacuation of pus in a chlorotic woman, who had had mastitis with formation of an abscess during the child-bed state.—Finally, it must not be forgotten that cataleptoid symptoms may be voluntarily simulated by both men and women, and that a rich field is here offered to skilful deception, which almost never fails in producing the desired result. Numerous old and recent examples show in this direction the most interesting characteristic proofs both of the persistence and routine of the deceiver and of the credulity of the deluded public.

Symptoms and Course.

The complete portraiture of catalepsy is composed of a succession of separate attacks, which are separated one from the other by a longer or shorter interval, often entirely without symptoms, often filled with complicated disturbances. The individual attacks themselves differ very much in intensity and extent, and show the pathognomonic symptoms of the cataleptic condition more or less well marked. On this account a distinction has been made between *catalepsia vera* and *catalepsia spuria.* The latter, indeed, includes very heterogeneous conditions, and is in part identical with the form called "trance" or "catalepsia hysterica;" in part also it includes the symptoms of ecstasy and somnambulism, without sharply defining the boundary. Since it is, however, essentially a manifestation of hysteria or of certain chronic cerebral affections, we must refer to the appropriate diseases for a description of the symptoms.

The attacks of catalepsia vera occur suddenly, as a rule; more rarely there are prodromal symptoms, consisting in an undefined sense of discomfort, headache, vertigo, trembling of individual muscles, hiccough, etc. Sometimes the patients stop suddenly in the middle of a sentence or of a motion, as if enchanted, with open mouth or raised arm, as the sudden stiffness seizes the muscle which is acting. In genuine catalepsy this peculiar condition of stiffness, or rather of extreme muscular tension, extends very rapidly over all the voluntary muscles, though not always over all in equal proportion; more rarely the stiffness is only partial or unilateral. The affected muscles have a firm feeling, and offer more or less resistance to passive motion. This resistance gradually ceases, and the muscles, though still contracted and firm, yet yield to passive motions, so that the limb can be placed in any desired position, in which it then again remains without change. It is this wonderful property which has received the name of wax-like flexibility (flexibilitas cerea). But there seems to be a great amount of exaggeration in the description of this condition, or, at least, in its highest degree it is one of the most rare phenomena.

Apart from the passive mobility of muscles apparently so stiff, the peculiarity of the condition is this: positions which normally —that is, under the influence of the will—either cannot be taken, or, on account of pain and weariness, can be maintained only temporarily, in catalepsy become persistent for a longer or shorter time—a phenomenon which may be explained by the more or less complete loss of consciousness, and probably in part by the reflex muscular tension (see "Theory of the Disease"). The phenomena just mentioned can be most easily produced in the upper extremities, and, indeed, more easily in those portions of these in which the joints are smaller and the surrounding mass of muscle is less, so that changes in the position of the thumb, the fingers, the hand, can be produced more easily than malposition of the shoulder or elbow. The same is true of the lower extremities; only, for analogous reasons, it is much more difficult to produce decided change of position than in corresponding divisions of the arms, and the attempt is much less successful; yet sometimes it is possible to produce very wonderful postures, not only of the extremities, but also of the whole body; as, for example, the patient can be placed in bed upon his pelvis, with his arms raised, and the legs directed upward at an acute angle, so that the body takes the shape of a V. Much that is reported in old authors concerning the wonderful positions of those possessed and the convulsed, concerning their soaring in the air, etc., is certainly to be referred to cataleptic muscular spasm. Moreover, the condition does not persist long in its full intensity; as a rule, after some minutes there is a diminution of the stiffness, so that the limbs obey the laws of gravity, and the arm raised horizontally sinks somewhat; the extended hand is slightly flexed; sometimes also a slight trembling gives notice of the slowly appearing exhaustion of the muscles.

The *consciousness* is frequently entirely lost from the commencement of the attack; the patients do not give the least sign of sensation or will, and later can remember nothing concerning the attack. In other cases, at least in the beginning of the attack, there is a dull consciousness, or such may be present during its whole duration. Then also, as a rule, there is not complete analgesia ; weak irritations are not noticed indeed, but stronger ones (as severe faradic metallic pencilling) may excite pain, and the patients afterwards remember the sensation. In one case reported by Puel it is said that even hyperæsthesia existed during the attack. The slightest touch, a light noise, caused gritting of the teeth, smothered cry, weak motions. Also in a case reported by Lasègue there was hyperæsthesia of the left half of the body, while on the right side, which was chiefly cataleptic, there was anæsthesia. The reflex irritability is quite variously affected; sometimes it is entirely lost; sometimes reflex actions are performed ; *e. g.*, the eyelids close on touching the conjunctiva ; the pupils react to light ; on the application of strong odors, the eyes water, the face flushes, and the pulse is accelerated. It is especially deserving of notice that a disturbance of consciousness may continue for a while after the cessation of the attack, and the ability of co-ordinating motions (*e. g.*, speech) may return very slowly.

In one case, during the attack, M. Rosenthal found the electrical reaction perfectly normal for both faradic and galvanic currents; in another case, however, the electro-muscular contractility, as well as the galvanic irritability of the nerve-trunk and plexus, was decidedly increased. In this case, where there was flexibilitas cerea, the position taken on account of the faradic irritation of the extensors or flexors of the arm, or as a result of the galvanization of the corresponding nerve, was not maintained after the cessation of the irritation; the hand returned to its former position. Benedikt also mentions a case with clearly marked flexibilitas cerea, where the galvanic nervous irritability (radial, median) was somewhat increased. The faradic irritability was found, sometimes by direct, sometimes also by indirect irritation, diminished in certain groups of muscles (interossei, quadriceps cruris, the flexors of the leg).[1]

As a rule, the general vital functions undergo no marked change during the attack. The respiration is generally of normal frequency, sometimes rather slow, more frequently of diminished or irregular intensity, so that lighter and deeper inspirations alternate. The pulse may also be slower, with slight excursion and diminished tension of the arteries. The temperature generally remains normal, but in certain cases is decidedly lowered; thus, in a case observed by Skoda, during the attacks the body was often very cold, and this coldness once continued forty-eight hours. Very severe cases, in which, with icy cold skin, the pulse and respiration became so weak as to be imperceptible, may have led the laity to consider the patients apparently or truly dead. Such cases form the foundation for the many exaggerated and wonderfully distorted stories in regard to cataleptic persons who have seemed to be dead during several days, or who have been buried alive. It is perfectly natural that the above phenomena, as also the wonderful peculiarities of the cataleptic condition, should always make a specially weird impression upon the laity, and thus give material for exaggeration and invention. But all such stories cannot be consigned unconditionally to the region of fable, as many medical authors are all too ready to do. The older stories, concerning apparent death and burying alive, are certainly so distorted and misrepresented that even in the most favorable case it is difficult to discover the grain of truth upon which they are founded. But more recently there are cases on record which show the possibility of such occurrences. Besides the above-mentioned case by Skoda, there is a similar report by Hasse, who saw a girl of very nervous disposition, twenty years old, lie about eight weeks entirely unconscious after jumping into the water, whose condition alternated between attacks of spasm and a cataleptic condition; in the paroxysms, which finally lasted nearly all day, the patient was deathly cold and seemed dead. Wilhelm also noticed in a girl eight years old, subject to somnambulism, that in the attacks, accompanied with flexibilitas cerea, there was a coolness of the skin and a livid color of the face (the urine also was alkaline during the attack).

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[1] Nervenpathologie und Elektrotherapie. 1874. p. 305.

The duration of the separate attacks is very variable: sometimes they continue only a few minutes, sometimes hours, even days; in the attacks described as so protracted, there are generally remissions or intermissions, in which the consciousness returns, the patients at least partially recover, then an internal or external irritation again gives rise to the attack. Strictly speaking, it is rather a cycle of attacks quickly following one another, as is seen in many severe neuralgias (prosopalgia, angina pectoris, etc.).—Sometimes the condition disappears quite suddenly; the patients recover at once full consciousness and the normal use of their muscles, take up their employment which had been interrupted, continue the sentence previously commenced, and conduct themselves as if not the slightest thing had intervened. Such cases are, however, exceptional. Much more frequently the patient's recovery is only slow and gradual; they are at first somewhat stupefied, as if just awaking from an unusually sound sleep. Sensibility is still diminished, the power of the will weakened; a certain amount of stiffness of the muscles still remains for some time, which renders motion difficult and slow.

In simple uncomplicated cases of catalepsy there are generally no symptoms in the intervals between the attacks, or they are limited to a certain irritability and lack of nervous harmony, sometimes also slight lassitude, more frequently vertigo and headache. It is indeed very doubtful whether these symptoms are to be considered as the result of the cataleptic attack, or as the manifestation of a neurotic diathesis. In simple catalepsy there is rarely any mental symptom noticeable during the intermission between attacks; the intelligence may even be remarkably developed (*e. g.*, in cataleptic children).—Of course, this is not the case where the catalepsy is merely one of a number of modes in which hysteria is manifested, or where there is a complication with other neuroses (insanity, epilepsy, chorea, etc.). Then there may be most varied symptoms, sometimes very severe, during the intermission, and to enumerate all such would require too much time; it is only necessary to mention as most frequent the occurrence of hysterical convulsions, delirium, maniacal attacks, hallucinations, and finally the combination with ecstasy and somnambulism.

The *course* of the disease is very irregular, but in by far the larger number of cases it is chronic. Those cases which seem to be caused by a malarial infection form almost the only exception in this respect; in such cases the attacks follow the regular course of other malarial neuroses (usually quotidian), are sometimes accompanied with fever and sweating, and yield to the usual antimalarial remedies. Occasionally a case which has as its cause an accidental injury (fright, injury) may be considered as ended after a few attacks occurring in rapid succession or even after a single attack. But generally the affection extends through many years, if not the entire life. Important individual peculiarities are caused by the frequency of the attacks, the duration of the intermissions, also by the presence or absence of complications.

As among epileptics, so among cataleptics there is a small number who, with a clearly marked predisposition, nevertheless suffer from only a small number of attacks, which are separated from each other by intervals of many years. Others, again, are found who have the attacks comparatively often, or periodically, now frequently, now rarely; still others, finally, in whom the slightest external influence is sufficient to excite a cataleptic attack at any time. The greatest susceptibility to irritation among the latter class is found in hysterical patients, and to these belong that condition described by Lasègue as "catalepsie passagère," where sleep, a cataleptic muscular stiffness, etc., can be produced by laying the hand on the eyes, or by closing the eyelids in any other way. Similar phenomena were observed by Lasègue also in many males who were emaciated and debilitated, or affected with uncertain cerebral symptoms.—A fatal termination is almost never caused by the cataleptic attacks themselves; but death is frequently the consequence of the original affection, or of some complication.

Anatomical Changes and Theory of the Disease.

The number of autopsies heretofore published is extremely small; they relate besides to such complicated diseased conditions, that they furnish no essential instruction as to the pathogenesis of catalepsy.

An examination reported by Schwartz relates to the case of the boy already referred to, who, after an injury, had at first attacks resembling chorea, later cataleptico-tetanic attacks, and after two years died from anæmia and marasmus. There was found in this case, besides a serous effusion in the arachnoid, a softening of the corpus striatum and optic thalamus, especially on the left side; along the posterior surface of the spinal cord, from the cervical to the lumbar enlargement, was a brownish-red, jelly-like mass, arranged in groups, covering the dura mater. The spinal cord seemed healthy. (There was no microscopic examination.)—Meissner examined a man, forty-seven years old, who had suffered from catalepsy for six years, and in the three last years also from maniacal and epileptic symptoms, with paralysis of the right side; he found in the anterior cerebral fossa over the ethmoid bone an epithelioma growing from the dura mater; the anterior third of the right cerebral hemisphere, as far as the cortex, was much softened, also the outer part of the right corpus striatum.—Lasègue found no change in the brains of two men affected with catalepsy.

If we examine more closely from a physiological point of view the combination of symptoms known as catalepsy, it is especially the cataleptic muscular tension (or rigidity) and the flexibilitas cerea which as pathognomonic attract the most attention. Most authors are inclined to the opinion that the cataleptic rigidity is only an increase of the normal tonus of the voluntary muscles, occurring occasionally in the attacks. However, the existence of this tonus is not accepted beyond all question; yet a small number of experimental facts tend to support the opinion that there is a reflex tonus depending probably upon the ganglion cells of the spinal cord. It may still remain doubtful whether the excessive increase of this normal muscular tonus causes a weakening or interruption in the conduction of the impulse of the will in the central organs, or, on the other hand, whether the diminution of the voluntary innervation causes an increase in the reflex tonus of the muscles. At all events, even when consciousness is not entirely lost, we always see the cataleptic muscular rigidity associated with diminution of the influence of the will in the affected muscles. Without the aid of a muscular tonus the phenomena of the cataleptic attack are difficult to explain. At least it is only a circumlocution without explanation to refer with Blasius to the general law of the stability of the nervous system, or with L. Meyer to a faculty of retaining a position after cessation of the voluntary innervation.

If—as seems enjoined by reasons to be yet considered—we look upon the cataleptic muscular contraction as reflex, without adopting the theory of a muscular tonus, we must imagine that in the cataleptic attack there are conditions especially favorable for the excitation of reflex action in the entire voluntary muscular system. The abolition of sensation and the influence of the will during the attack, the complete loss of consciousness generally noticed, show that there is a temporary complete or incomplete cessation of cerebral function. The removal of the cerebral volition and the entire psycho-motor action renders it more easy for the occurrence of reflex muscular action, which is often prevented by the will. But besides, possibly at the same time, those reflex inhibitory centres discovered by Setschenow, situated in the corpora quadrigemina and optic thalamus (in frogs), and possibly in the upper part of the medulla oblongata, are thrown out of action. Though this view can be received only with reserve as purely hypothetical, yet one fact rather tends to give it some support, namely: the observation often made that narcotics and anæsthetics, at a certain stage of their action, before the production of narcotism, may give rise to slight epileptic phenomena.[1] From experiments made by Malkiewicz under the direction of Setschenow, we know that a number of toxic substances, among them alcohol and morphia, exert a decidedly paralyzing influence upon the cerebral reflex inhibiting centres, and it is rational to refer the cataleptic muscular contraction occurring from the use of narcotics and anæsthetics, as well as the reflex spasms sometimes seen during opium poisoning, to diminution or absence of the function of the cerebral inhibitory organs. Certainly, hereby only one link in the chain of the cataleptic phenomena is explained, and by no means is a special explanation of the cataleptic condition given, but only the common preliminary condition of such an explanation is furnished.

The explanation of the "flexibilitas cerea" is perhaps still more difficult than that of the cataleptic muscular rigidity. This difficulty does not, however, lie in the point where one is most accustomed to look for it; namely, that unusual or unnatural positions can be assumed and maintained for a long time. The possibility of such positions is much more simply explained by the absence of volition with persisting reflex action and co-ordination, *i. e.*, with unrestrained or even exaggerated function of the reflex apparatus and the co-ordinating centres of the spinal cord and medulla oblongata. The integrity of these centres renders it apparently possible that in the cataleptic attacks the balance can be maintained in such abnormal positions as the above described V-form, to which normally the influence of the will would be opposed. That it is truly the influence of the will which (consciously or unconsciously) exclusively or chiefly hinders or inhibits the taking of such postures is seen according to my view very convincingly in the simple fact that skilful pretenders can imitate the cataleptic phenomena—even the abnormal positions of the limbs—so as to completely deceive. Here the will not only does not oppose such co-ordinations as are usually resisted, because painful, tiring or useless, but, on the contrary, it is specially directed to produce them, and is very strongly interested in maintaining them.—Hence, the chief difficulty does not lie in this direction; but rather in the great instability of the position taken notwithstanding the apparent tendency to stability; in the circumstance that an impulse from without, a slight pull or push, etc., immediately gives the limb a new position, calls into play a new combination of the reflex and co-ordinating apparatus, which again remains fixed until another impulse is received. The most reasonable, and on many accounts most evident, explanation is that the passive motion by means of the sensory muscular nerves acts reflexively upon the motor centre (or the co-ordinating centre of the spinal cord). Nevertheless, many considerations opposed to this occur to us; first, the fact that the reflex irritability in catalepsy is not increased, but frequently seems diminished or abolished. Doubts may perhaps be raised as to this by the supposition of a different relation of the sensory muscular nerves and the sensory cutaneous nerves as well as of the centres of co-ordination and other reflex apparatus. But upon what does it depend that, while a passive motion easily causes a change in the position of the limbs, which is permanent, the most powerful faradic or galvanic irritation of muscles or nerves does not have the same effect? (See "Symptoms.") Yet, in the latter case there is certainly a much more intense irritation of the sensory muscular nerves than can be caused by moderate passive extension or shortening!—Benedikt remarks in regard to the flexibilitas cerea: "Since every muscle by every extension loses as much power as it gains by contraction, it is clear that when once antagonistic muscles have that tension which is suitable to maintain a portion of the body in a certain position, the same tension is sufficient for all positions in the direction of those muscles." This remark is correct, but yet cannot answer the above-mentioned contradiction. Rosenthal also only states more precisely the same thing, but does not explain it, when he says: "While therefore the irritation, acting directly upon the skin or electrical irritation of muscles or nerves, gives rise to no reflex action, at least which may cause the position artificially produced to be permanent, the indirect irritation of external influence can be reflected from the sensory nerves, especially of the bones and the joints, upon certain groups of muscles. The so-called waxy mobility is then a reflex contraction."—To the latter view we are at all events driven; but just the how? and wherefore? of the form of reflex action is, alas! still unknown to us.

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[1] I have myself seen an exquisite case of flexibilitas cerea, alternating with trismus, opisthotonos, and general convulsions, in a patient poisoned by morphia (by 0.09 gramme [a grain and one-third] of hydrochlorate of morphia).

The special analysis of the remaining secondary and subordinate phenomena of the cataleptic attack must under these circumstances be of little value; what we may know or imagine, is for the most part deducible from that which has already been said.

Diagnosis, Prognosis, and Treatment.

In well-marked cases of catalepsy there is no difficulty in diagnosis, if by that term is understood a combination of symptoms characterized especially by muscular stiffening and the flexibilitas cerea. Conditions wherein these pathognomonic phenomena do not occur are then not catalepsy, or at least not catalepsia vera, and may well be included in the large and quite indefinite category of catalepsia spuria, or, according to their special characteristics, among the choreic, epileptic, hysterical forms of spasms. Only a persistent and cleverly executed simulation could give rise to diagnostic difficulty, or at least to doubt. That such a simulation is possible is shown by different examples from ancient and modern records (among others, the cases from Corlieu and Walcher often referred to in writings). Prolonged observation, more careful testing of the sensibility and reflex irritability, perhaps, too, the examination of the electrical reactions, would, as a rule, afford a safeguard against deception.

The *prognosis* of catalepsy, so far as concerns life, is favorable, if we except complications; so far as concerns the disease, it is generally unfavorable, since in the majority of cases a spontaneous recovery, or one due to treatment, is not at all probable. Yet, as we have seen, there are exceptions; especially do those cases, caused by a malarial infection, running a typical course, furnish a better prognosis; the same is true in regard to many cases of catalepsy occurring in relatively healthy persons from more accidental causes (injuries, mental shocks, etc.) In inveterate cases, with nervous predisposition and severe complications (hysteria, insanity, etc.), the prognosis is, of course, especially unfavorable.

Two problems may be proposed in the *treatment:* first, to stop the attack, or cut it short; then, to prevent the return of the attacks. The latter task can be undertaken with confidence of success only where there are grounds for believing that the cause can be removed, as in malarial infection. In such cases improvement or recovery has been obtained by the use of quinia, as is shown by the reports of Medicus, Eisenmann, Glas, and others. In other cases quinia alone or combined with morphia (Hartmann) has been of benefit. Favorable results have been obtained also by the use of tonics, iron, ergot (Glas), morphia and the cold douche (Smoler), and by electricity in the form of the induced current (Macedo). The constant current has heretofore given no good result (Benedikt, Rosenthal). Rosenthal also found in one case that the subcutaneous injection of curare had no marked influence.

As to whether a palliative treatment of the attack is to be recommended or not, cannot be decided off-hand; at all events, the danger of interfering, claimed by one party, is not clearly proven—but also, certainly, the efforts heretofore made have given few positive results. In one case of Lasègue's a bleeding during the attack had no influence upon the cataleptic condition. Calvi thinks that by the injection of tartrate of antimony into a vein of the arm he caused the cataleptic rigidity to disappear. Jones recommends for the same purpose wet packing, cold douche, and the induction current. In the hysterical cases designated by Lasègue, "catalepsie passagère," the patients awake from their somnolence sometimes under slight external irritation, *e. g.*, from sprinkling with water; but in true catalepsy a very strong cutaneous irritation has often no influence.—The use of such palliative remedies should be especially advised in attacks which last very long, as well as in the rarer cases which are accompanied with more severe disturbances of the circulation and respiration, great weakness of the cardiac action—a condition simulating death. In very protracted cases or in those attacks which consist of a series of attacks rapidly following each other, separated by imperfect remissions, lasting through many days and weeks, artificial feeding by the stomach-tube or by nutritious enemata may also be necessary.

TREMOR.

(Trembling.)

v. Swieten, Commentaria. t. II. p. 167. Paris, 1771.—*Mérat*, Traité de la colique métallique. 2d. ed. Paris, 1812.—*Tanquerel des Planches*, Traité des maladies de plomb. Paris, 1839.—*Romberg*, Nervenkrankheiten. 2. Aufl. 1851. Bd. II.—*Brown-Séquard*, Experimental researches applied to physiology and pathology. New York, 1853.—*Schiff*, Lehrbuch der Physiologie. 1859. Bd. I.—*Gubler*, Arch. gén. de méd. 5. sér. XV. 1860. p. 702.—*Valentin*, Versuch einer physiologischen Pathologie der Nerven. Leipzig und Heidelberg, 1864. 2. Abth.—*Sanders*, Edinb. med. Journal. 1865, May.—*Ferrand*, Union médicale. 1868. No. 62; No. 107.—*Guéneau de Mussy*, Gaz. des hôp. 1868. Nos. 48, 50.—*A. Eulenburg*, Zur Therapie des Tremor. Berl. klin. Wochenschrift. 1872. No. 46.—*Cazenave*, Gaz. méd. de Paris. 1872. Nos. 18, 19, 27.—*Oulmont*, Bull. gén. de thér. 15. Dec. 1872.

By tremor or tremor musculorum (muscular trembling) is understood *a clonic spasm of the voluntary muscles, consisting in slight oscillatory contractions following each other in rhythmical order.* Tremor is therefore only a symptom, which is found, with others, locally in neuritis, more extensively also in certain central neuroses (disseminated sclerosis of the nerve-centres; paralysis agitans), and in chronic poisoning (alcohol, opium, lead, mercurial poisoning); but it may also appear under some circumstances as an independent disease, or at least as the only noticeable symptom (*tremor essentialis s. simplex*)—as a rule, with a very chronic course and gradual development.

The synonyms are: Astasie musculaire (Gubler); dysteria agitans (Sanders).

Etiology.

Simple uncomplicated tremor occurs especially in advanced age (*tremor senilis*). Those forms which depend upon chronic disease of the brain and spinal cord, as also toxical tremor, are, on the other hand, not dependent upon any particular time of life, and are observed most frequently in middle age, or, indeed, even in youth. However, simple tremor may exceptionally occur in persons who are still young, so that the terms "tremor simplex" and "tremor senilis" do not exactly coincide.

I have not noticed that women (as Hasse states) are more disposed to tremor than men. Though tremor may occur as one of the manifestations of hysteria, yet this is not very frequent.

Toxic tremor depends specially upon those conditions which lie at the foundation of the corresponding chronic poisoning; *tremor mercurialis* and *saturninus* are especially professional diseases: the former is found especially among gilders, looking-glass manufacturers, and artisans in quicksilver; the latter, among hat makers, and others, who are exposed to the vapors of lead. Yet, tremor mercurialis may arise from medicinal poisoning. The mercurial tremor is relatively much more frequent and characteristic than the saturnine.—The tremor caused by alcoholic intoxication is seen exclusively among drinkers (*tremor potatorum*)—that which depends upon opium, among opium-eaters. —Also chronic poisoning by nicotine, or the other injurious substances found in tobacco and tobacco smoke, seems to cause tremor. I have repeatedly met this symptom among young cigar makers who smoked to excess. Also the immoderate use of coffee and tea, further the prolonged use of small doses of quinia, so it seems, may give rise to tremor in some cases.

As causative may also be mentioned exhausting diseases (especially typhoid), great bodily and mental exertions, excess in venery, onanism. Trembling occurs temporarily in diseases accompanied with fever, especially during the chill. Sometimes tremor has been seen in young persons immediately after the receipt of an injury (fall upon the back, a thorn under a toe nail, Sanders). Frequently the cause of habitual tremor cannot be discovered.

Symptomatology and Course.

Apart from the various complications, muscular trembling, even in simple tremor, shows a great difference in intensity and extent. In the beginning, and in slight cases of the affection, the trembling is noticed, as a rule, generally or exclusively when voluntary motions are attempted, and then affects especially those muscles whose action is intended, or neighboring synergetic, or antagonistic muscles (muscles of the hand and arm in raising a weight or on attempting to write). Yet often also distant groups of muscles are immediately or gradually affected, whereby sometimes the extension seems to follow the laws of reflex action, as the symmetrical muscles of the opposite side are first called into action, then the muscles of the upper part of the body (*e. g.*, the muscles of the head when the arm is moved); later, and generally with much less strength, also the muscles of the lower limbs. The origin of the tremor has, in such cases, much similarity with choreic movements; yet, herein is a difference, that the trembling up to a certain degree can be suppressed or moderated by increased force of will, while strong effort of the will in chorea generally causes an increase of the tumultuous motions. Also the perfect concentration of the attention, especially strongly exercised mental power, may sometimes temporarily cause a cessation or remission of the muscular trembling. On the other hand, this often disappears when there is more complete bodily and mental rest, with mechanical support of the trembling limb, while reposing in a horizontal position, and almost always during sleep. In a similar way the tremor of drunkards and opium-eaters may be made to disappear temporarily by renewing the use of alcohol or opium.

In simple tremor, trembling rarely reaches that degree of intensity which is seen in that variety which is accompanied with paralysis, as may be seen in its prototype—paralysis agitans (see the following section). Hence, we generally notice only scintillating oscillating vibrations of separate groups of muscles; the affection rarely reaches such severity as to show spasmodic contraction of all the muscles, and thus resemble shaking palsy. The power of the affected muscles, so soon as the trembling ceases, is entirely normal; and even while the trembling persists it is not much diminished. The electrical (faradic and galvanic) reaction of the agitated muscles shows no change. Sensibility and reflex irritability are also not affected.

All the voluntary muscles of the body may be affected by tremor; yet the affection much the most frequently begins in the arm, and extends then in the manner already mentioned; more rarely the muscles which move the head, or those of the lower limbs, are first attacked. The facial muscles of expression, the muscles of mastication, and those of the tongue possess, indeed, no immunity, but in simple tremor are very seldom affected, but in toxic and febrile tremor they are frequently implicated. (The irregularity of the heart's action, known as "tremor cordis," occurs under quite different conditions, and presents no precise points of comparison.)

The duration of tremor is generally exceedingly protracted. The affection in most cases continually increases in severity, so that the trembling occurs no more merely when a motion is intended and upon special occasions, but (at least during the day) persists continuously with variations of exacerbations and remissions, with, at most, short intervals of quiet. Only exceptionally, in chronic trembling, which has become habitual, is there seen spontaneous improvement or recovery. Of course, however, cases of tremor may occur—especially in young persons and where the affection is due to accidental injuries (*e. g.*, wounds)—which have a favorable course, as the symptom, after a relatively short duration, ceases spontaneously or after a seemingly insignificant treatment.

Among the various forms of toxic tremor, tremor mercurialis presents a combination of symptoms which have the greatest resemblance to paralysis agitans, and hence, by some authors (Copland, for instance) are considered completely identical therewith. This similarity relates as well to the intensity and the extent of the tremor itself as to secondary paretic symptoms and the accompanying cerebral, especially psychical symptoms (see "Paralysis Agitans").—In tremor saturninus there have usually been noticed long previously other symptoms of chronic saturnine intoxication (lead colic, lead paralysis, often also arthralgia, etc.); the trembling, moreover, much more rarely extends to the whole muscular structure; as a rule, it is rather limited to the upper extremities and to individual facial muscles (orbicularis, levator anguli oris); again, there may be added to the tremor many other disturbances of the nervous system—paralysis, anæsthesia, amaurosis, and the combination of the so-called encephalopathia saturnina: delirium, coma, epileptiform convulsions, etc.—Likewise only slight resemblance with simple tremor is seen in the trembling which occurs in chronic alcoholism, and which forms one of the most ordinary symptoms of this condition. Here also the trembling begins usually in the hands, extends gradually to the arms, body, legs, even to the lips and tongue, and may increase finally even to a shaking of the body, so as to interfere with standing or walking. The tremor potatorum is strongest during fasting, especially in the morning, and is less after the use of spirit. Further, progressive muscular weakness and paralysis may set in, as well as numerous nervous disturbances: creeping sensations, arthralgia, anæsthesia, clonic and epileptiform convulsions, and the combined phenomena of delirium tremens. Besides, there are usually present previously the disturbances excited in other organs by the abuse of spirit, especially in the digestive organs.—In the tremor of opium-eaters are seen at the same time other symptoms of chronic opium poisoning (contracted pupil, constipation, etc.). The tremor which depends upon the action of nicotine may sometimes be unilateral, or at least affect one-half of the body more than the other, yet it is generally accompanied with various nervous disturbances (muscular weakness, dizziness, neuralgia, myosis, etc.).

Theory of the Disease.

In the general consideration of tremor, two questions arise which are of great interest theoretically: what sort of change or irritation of the motor nervous elements, what specific mode of irritation, excites the anomalous reaction which is termed tremor?—and what portions of the nervous system are the seat of its action, the point attacked by the irritation?—From the lack of pathological facts, we must depend upon the results of experimental physiology, which here also does not fail us. As Schiff first discovered, the phenomenon of trembling is seen frequently in muscles which are experimentally deprived of the influence of the will. This is most strikingly seen in the muscles of the tongue in dogs after section of the hypoglossus. The vibratory motion of the bundles of muscular fibres are clearly seen through the overlying mucous tissue, and, when the section is on one side only, this is seen only on the paralyzed side, and can be noticed as well when the tongue is protruded as when it rests quietly within the mouth. When the contraction of one muscular bundle ceases, that of a neighboring bundle begins; but there is no regular extension of the trembling, and the organ is not moved thereby. Similarly, in rabbits, after section of the facial, there is a continual oscillation of the whiskers; in birds, after section of the motor oculi, there is trembling of the iris (which is provided with striated fibres). Likewise, trembling is seen in the muscles of a limb when laid bare, whose nerves have been separated from their centre. Yet, this phenomenon does not appear immediately after the separation, but first some days later; towards the end of the first week, it gradually reaches its maximum, and then may continue months or even years. It forms therefore only a secondary effect of nerve section, and is probably to be considered as the consequence of the progressing centrifugal degeneration of the peripheral portion of the nerve. As the well-known recent investigations of Erb, Ziemssen, and Weiss have shown, this causes consecutive changes in the muscles (in the form of interstitial myositis), which perhaps may also favor the existence of tremor under the above-mentioned conditions.

Pathological observations in men offer many points of resemblance with these experimental data, in so far as that we see tremor occur as a common symptom in cases where the influence of the will has ceased or has become weakened, often locally in paralyzed or atrophied muscles. Besides the trembling in neuritis, in paralysis agitans, in sclerosis of the nerve centres, etc., here belongs also the trembling found in nervous persons, the energy of whose will is temporarily or permanently affected (*e. g.*, hysterical persons), and in the aged, who, in consequence of general disturbance of nutrition, have no longer the normal control over their muscles; the trembling in the chill of fever, after exhausting disease, etc., as well as, finally, the tremor which depends upon the influence of poisonous substances which may act upon the functioning of the central or peripheral nervous system, in part irritating, or in part paralyzing. Under all circumstances there is presented to us a condition of the motor apparatus depending upon disturbances of the voluntary innervation, from nutritive changes in the central or in the peripheral portion of the motor nervous system. While the influence of the will upon the motor centre is diminished, there is an increased irritability of the motor nervous elements in all other regions of the motor apparatus, or in some single region, in consequence of which the slightest irritations which are physiologically inoperative (*e. g.*, the usual circulatory and nutritive processes) give rise to that pathognomonic anomaly in the muscular reaction. Hence, there was undoubtedly some justification for placing tremor midway between akinesia and hyperkinesia, for allowing it (in the words of Romberg) "to form the bridge from spasm to paralysis." On the other hand, it seems so much the less necessary to divide trembling into two varieties—a paralytic and a convulsive—as is done by van Swieten, Gubler, Charcot, Ferrand, especially if the convulsive form depends (according to Ferrand) upon a weakness of the muscular tone.

In reference to the point whence general tremulous motions arise, experiment has not heretofore given decisive answer. Earlier investigations by Magendie, Volkmann, and others, point to the spinal cord; thus, B. Volkmann mentions that trembling can be experimentally produced by subjecting the spinal cord of a decapitated animal to the influence of a weak current from a magneto-electrical apparatus, and turning the wheel rather slowly.[1] Onimus, again, refers trembling to the cerebellum, as the common organ of muscular tone. He saw in ducks, after destruction of a small portion of the cerebellum, once also when a clot of blood pressed upon the posterior part of the cerebellum, a persistent rhythmic trembling of the head, especially motions of the bill. The question of the cerebral or spinal origin of tremor will come up again when we consider paralysis agitans. Ferrand locates the point of origin of "convulsive" trembling in the spinal cord, in its gray substance, which, according to Vulpian, regulates the muscular tone as a reflex act. Sanders also seeks to refer the cause of what he calls "dysteria agitans," which agrees essentially with simple tremor, to an increased irritability of the motor centres of the spinal cord, which can be referred to an anæmia of its gray substance. The supposition of a spinal origin of simple tremor is strengthened also by the fact that usually the muscles innervated by the cranial nerves are spared (see above). On the other hand, other forms of tremor, in which the facial muscles of expression, the muscles of mastication, the muscles of the tongue, are frequently affected, are probably of cerebral origin. This is especially true of mercurial and alcoholic tremor, which can be referred to a cerebral origin, from the accompanying paralytic or other concomitant symptoms, especially psychical; possibly the same is true of the tremor caused by opium, tobacco, quinia; also of that from fever.

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[1] Article "Nervenphysiologie" in *B. Wagner's* Handwörterbuch der Physiologie. p. 488.

Treatment.

Of course, in all those cases wherein the cause can be discovered and removed, the treatment of tremor must first of all be causal. This is especially true in cases of tremor caused by poison, by the injurious influence of occupation, the abuse of liquor, or other indulgence. In the treatment of mercurial and saturnine tremor the elimination of the poison must be favored as much as possible. For this purpose (besides the means of simply increasing the secretions) iodide of potassium, sulphur baths, and other preparations of sulphur are held in especial, though scarcely justifiable, esteem.

In those forms of tremor which are not amenable to causal treatment, a large number of the most different and in part heroic means have been used empirically—generally, indeed, with most insignificant results. However, the tonics (ferruginous preparations and waters, quinia) have obtained the most general recommendation, also residence at high altitudes, and use of hot spring baths (Ragatz-Pfaefers). According to my experience these remedies, at least in the severe and old cases, are insufficient. Also, I have never seen any results worth mentioning from the use of narcotics (morphia, chloral, calabar bean, curare, etc.), or of bromide of potassium. The best results comparatively have been obtained by me in simple tremor by the methodical use of subcutaneous injections of arsenic. (Fowler's solution diluted with two parts of distilled water—of which from six to ten minims,—about half of an ordinary hypodermic injection syringe,—is to be given for a dose.) These injections can be continued daily for a long time without the appearance of toxical or other unpleasant symptoms; they have this especial advantage over the internal use of arsenic, that the disturbance of the digestion caused by the latter does not occur, and they are more certain in their action. Holst and others (according to oral communication) have also obtained favorable results in tremor by the injection of arsenic.

Of the internal remedies otherwise recommended may be mentioned, among others, hyoscyamin, from which Oulmont has obtained very marked effects in tremor mercurialis, as also in two cases with severe senile tremor. I have used the hyoscyamin up to one-twentieth of a grain daily for a long time without effect in a very severe and old case of tremor of probably alcoholic origin, which had obstinately resisted the arsenic injections.—In mercurial tremor, Guéneau de Mussy recommended the phosphide of zinc (one-half grain daily, given in pill-form); Lafon and Gardanne saw benefit from electricity; Brockmann from the cold water douche with sulphur baths, and the internal use of nervines. Huss has recommended fusel-oil (amylic alcohol) in tremor potatorum.

As palliative, especially in extreme trembling of the hand, mechanical appliances can be used to fix the hand, and so render writing possible. Cazenave, among others, has lately contrived a special apparatus for this.

PARALYSIS AGITANS.

(Shaking Palsy—The Trembles.)

Parkinson, Essay on the shaking palsy. London, 1817.—*Todd*, Art. Paralysis in Cyclopædia of pract. med.—*Romberg*, Nervenkrankheiten. 2. Aufl. 1851. Bd. II.—*Blasius*, Stabilitätsneurosen. Archiv d. phys. Heilk. 1851. S. 225.—*Basedow*, Casper's Wochenschrift. 1851. No. 33.—*Remak*, Galvanotherapie. Berlin, 1858.—*Reynolds*, Lancet. 3. Dec. 1859.—*Cohn*, Wiener med. Wochenschrift. 1860. No. 18. ff.—*Skoda*, Wien. Med. Halle, 1862. 13.—*Charcot* and *Vulpian*, Gaz. Hebd. 1861. p. 765 and 816; 1862. p. 54.—*Topinard*, Gaz. des Hôp. 1866. No. 21, 46.—*Ordenstein*, Sur la paralysie agitante et la sclérose en plaques, thèse. Paris, 1867.—*Sanders* in Reynolds' System of Medicine. Vol. II. London, 1868.—*Villemin*, Rec. de mém. de méd. etc. milit. 3. sér. XXV. p. 116. Aug., 1870.—*Handfield Jones*, Studies on functional nervous diseases. London, 1870.—*Ball*, Med. Times and Gaz. 1. Oct., 1870. p. 385.—*Bourillon*, Gaz. des Hôp. 1870. No. 50, 51.—*Betz*, Memorab. No. 3. 1870.—*Althaus*, Med. Times and Gaz. 1870. II. p. 643.—*Meredith Clymer*, Lectures on the palsies and kindred disorders of the nervous system. Med. Record. 1870.—*A. Eulenburg*, Lehrbuch der functionellen Nervenkrankheiten. Berlin, 1871.—*Meschede*, Virchow's Archiv. Bd. L. Heft 2. 1870.—*Chvostek*, Wiener med. Wochenschrift. 1871. No. 37-39.—*Joffroy*, Gaz. des Hôp. 1871. No. 151.—*Murchison* and *Cayley*, Transact. of the path. soc. XXII. p. 24. 1871.—*Joffroy*, Arch. de phys. norm. et path. 1872. No. 1.—*Claveleira*, De la paralysie agitante. 1872.—*Kuehne*, Inaug, Diss. Berlin, 1872.—*A. Eulenburg*, Berl. klin. Wochenschrift. 1872. No. 46.—*Charcot*, Klinische Vorträge über die Krankheiten des Nervensystems. Deutsch von Fetzer. Stuttgart, 1874.—*Benedikt*, Nervenpathologie and Elektrotherapie. Leipzig, 1874.

By *shaking palsy* or *paralysis agitans* is understood a combination of symptoms, the chief elements of which, as its name implies, are two phenomena: a gradually increasing motor weakness and a trembling in the voluntary muscles of the body, the latter, however, preceding the paralytic symptoms, or at least being present in a greater degree.

As Galen already distinguished two kinds of trembling (παλμος and τρεμος), and van Swieten a "tremor a debilitate" and "tremor coactus," it seems to have been a more or less complete differentiation of paralysis agitans, at least of its allied disorders, and simple tremor. Yet Parkinson (1817) was clearly the first to accurately describe the disease. Nevertheless it was afterwards many times confounded in part with chorea, in part with other forms of trembling, finally, in part with an affection only recently fully recognized—multiple or disseminated sclerosis of the nerve-centres (sclérose en plaques disséminées). While Trousseau and Sée, in their work on chorea and other diseases, cleared up also the distinction between this disease and paralysis agitans, the distinction between the latter and multiple sclerosis, which was more difficult to settle, was shown by Charcot and Ordenstein (1867). A succession of works concerning multiple sclerosis, which have appeared since then (by Bourneville, Leo, Baerwinkel, Schuele, Liouville, Leube, Erbstein, Jolly, and others) as well as some new post-mortem examinations in paralysis agitans, have assisted materially in more exactly distinguishing the two diseases one from the other. Yet the number of pathologico-anatomical examinations is still very small, and is not at all sufficient to justify the forming of a satisfactory theory of the clinical combination of symptoms in paralysis agitans.

Synonymous terms: Chorea festinans (Sauvages); chorea procursiva (Bernt); sclerotynbe festinans, etc.

Etiology.

Paralysis agitans is a disease which occurs at a relatively advanced age. It is rarely seen below forty, is most frequent after sixty years. Its appearance in childhood is most uncommon; yet Meschede mentions a case where the disease showed itself in a boy twelve years old (it is said after being kicked in the face by a horse). Duchenne observed a case where the affection occurred in the twentieth year. Men are much more frequently attacked than women. Special predisposing causes can scarcely be proved. One must, however, consider as such, age, particularly the senile condition of the heart and of the blood-vessels connected therewith. Yet organic disease of the heart or arterial sclerosis have not by any means been found in all cases of paralysis agitans during life, or even at the autopsy, when such has been made. Hereditary influences have hitherto, at least, not been proved to exist. In England and North America the affection should be especially frequent, yet it belongs also even there to the more rare affections. (According to Sanders there are in England and Wales on an average twenty-two fatal cases of paralysis agitans yearly, of which fourteen are males, eight females.)

The affection seems to occur chiefly in persons in the lower stations in life, who are found in unhealthy situations, have worked hard, do not inherit robust constitutions, and also have been subject to violent emotions. Many observers mention the effect of violent emotions, especially a sudden fright: *e. g.*, one by van Swieten of a man who was awakened by a frightful clap of thunder; another by Oppolzer of a man who was frightened by the bursting of a bomb at his side; a third by Hillairet of a man who saw his son murdered before his eyes. In all these cases the outbreak of the trembling followed immediately the exciting emotion. In other cases a wound is mentioned as the direct cause, *e. g.*, in the case of Meschede, already referred to, also in many cases reported by Sanders and Charcot, which, however, according to their descriptions, seem to belong to simple tremor rather than to paralysis agitans. In one case by Betz cold bathing of the head and hands, with a cold drink, was mentioned as the cause.

Symptoms and Course.

The initial and prominent symptom of the disease, the tremor, appears now as a peculiar trembling, in the form of weak, oscillating, rapidly repeated vibrations; now in strong clonic spasms, consisting of contractions, which occur by fits and starts (shaking palsy). These motions begin generally in the upper extremities, especially the hand and forearm, and gradually invade also the lower limbs, and also the facial muscles. In the latter case there is sometimes also disturbance of articulation (stuttering). More rarely the muscles of the body are implicated, among these especially the muscles of the neck, so that there is trembling or pendulum-like motion of the head. In individual cases the tremor remains confined to one-half of the body, or, indeed, to a single extremity (generally an upper). As a rule, both sides of the body, though in unequal degree, are successively attacked. In the same patient the trembling does not have always the same intensity and extent; sometimes it continues with undiminished severity for days; sometimes there are remissions lasting many days, or it temporarily leaves one limb, or a muscle, while it appears in others with greater severity. When the patient lies down, it is generally weaker or ceases entirely. In other cases the trembling is, indeed, never entirely wanting, but paroxysmally it increases to a great intensity, and, indeed, the occurrence of such paroxysms is clearly favored by bodily or mental exertion. But in general the trembling is completely independent of voluntary motor impulses as well as of passive motions, and this circumstance is of great value as a means of diagnosis to distinguish it from many forms of chorea and also from the trembling in disseminated sclerosis of the nervous centres.

The paralysis occurs generally secondarily after a longer or shorter persistence of the tremor, and gradually increases in intensity and extent, yet it almost always remains partial, and not rarely is more circumscribed or is irregularly developed in different muscles or muscular groups. As in various other paralyses, the extensors of the limbs are affected by preference, while on the other hand a corresponding stiffness appears in the flexors. In many cases there is no diminution in the motor power (measured, for example, with a dynamometer); again, a marked delay is seen in executing an intended motion, as in speaking, combined with a disproportionate weariness (Charcot). The electrical reaction for the induced as well as the galvanic current is entirely normal in the affected muscles, as I have convinced myself in some very old and severe cases. The trembling often abates in all the muscles, or in those especially implicated, while the paralysis increases. It is quite exceptional for the involuntary muscles of the bladder and the rectum to be affected by the paralysis.

In many cases there is developed gradually in the muscles of the limbs, also of the trunk and neck, a condition of excessive stiffness, recalling the rigidity of catalepsy, which, at first temporary, becomes later permanent, and may give rise to deformities. By the rigidity of the anterior cervical muscles the head is bent strongly forward (as Parkinson remarked), and can be raised or rotated only with difficulty. The body is also inclined forward, which causes the appearance of a tendency to fall forward noticeable in walking. The elbows usually stand somewhat off from the thorax; the forearm is slightly flexed on the arm; the fingers are semi-flexed, inclined towards the ulnar border of the hand, and in the various joints alternately flexed and extended (as is seen in arthritis deformans); thumb and index fingers are frequently extended and approached one to the other as in the position for writing. Similar deformities are developed in the lower extremities by the predominant contraction of individual muscles, especially of the muscles of the calf: the knees are adducted; thighs and feet are in the well-known position of equino-varus. Of course, the difficulty of performing any motion, especially locomotion, is greatly increased by this muscular contraction and the rigidity, which finally becomes permanent; the patients in walking give the impression as if they moved forward drawn up together, as if their joints were grown together (Charcot). Moreover, the advent of the stiffness occurs usually in an advanced stage of the affection; it is only exceptionally noticed in the beginning.—Another more uncommon phenomenon is the inclination to certain forms of compulsory motions, especially to a tendency to run forward. The patients, with difficulty and slowly, begin to walk, but then are obliged, without stopping, to move quickly forward; hence, they easily fall forward. This tendency to run forward has been explained by the fact that from the inclination of the head and body forward the centre of gravity has been displaced; however, this explanation is not satisfactory, or at least is not applicable to all cases, since in certain cases the opposite form of compulsory motion, the tendency to move backward, is noticed. In a patient, who had otherwise the impulse to move forward, Charcot could excite the impulse to move backward by lightly pulling his coat when he was standing still; then immediately there was a propulsion backward which soon became very impetuous, and the patient was in danger of falling backward if precaution was not taken to prevent it.—Meschede noticed in the case already quoted the propulsion forward to occur at the same time with an attack of laughter in a patient with very limited mental powers. In a case described by Topinard there was propulsion forward besides change of countenance, satyriasis and glycosuria; the latter symptom was improved by the use of Vichy water, while the others increased.

The *temperature* of the body measured in the axilla or in the rectum, notwithstanding the unremitting muscular action, *is not increased* in paralysis agitans, as is shown by the older observations (Charcot and Bouchard,[1] Ordenstein) as well as by my own observations. Also in many cases which I have repeatedly examined the temperature of the external auditory canal has not deviated from the normal.—Charcot explains the absence of increased temperature thus: it is found only in "static" (*i. e.*, predominating tonic), not in "dynamic" (predominating clonic) spasms—in tetanus, for example—but not in chorea and paralysis agitans.—Nevertheless, although the temperature of the body is normal, there is often a subjective sensation of increased warmth, especially after a paroxysm of trembling, sometimes also in connection with copious secretion of perspiration.

Apart from this sensation of excessive warmth, *disturbances of sensation* are noticed, now in the form of disagreeable sensations (crawling and pricking in the hands and feet), now in the form of partial, generally incomplete anæsthesia—yet they may be entirely wanting even in advanced cases of paralysis agitans. Functional disturbance of the nerves of special sense (optic, acoustic) are rarely noticed. But other cerebral symptoms of the most varied kind are rather frequent: headache, vertigo, sleeplessness, psychical disturbance, hypochondria. In a few cases there is well-marked melancholia; in other cases there are hallucinations, and maniacal attacks are noticed. Not rarely after the affection has continued rather long, there is a weakness of memory and of judgment, generally a dulling of the entire mental functions.

The *course* of the disease is generally very protracted, its duration, at least many years, sometimes even thirty years.

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[1] Mémoires de la soc. de biol. 1866.

Generally after the above-mentioned symptoms have continued a long time, there occurs increasing exhaustion of the nervous system, diminution of power, and the patient, almost immovable, has bed-sores, which finally cause death. It is deserving of notice that sometimes, a few days before death, the trembling entirely disappears (Claveleira, Lebert). In other cases death results from some intercurrent disease, especially pneumonia, pleurisy, variola, typhus.—Improvement and even recovery have been noticed by individual authors. Yet there is reason for the supposition that in the cases of pretended recovery the disease was not paralysis agitans. This supposition is strengthened when it is noticed how many authors, especially English authors, confound this affection with choreic conditions. Handfield Jones believes it must be admitted that there is a double form of paralysis agitans; one entirely incurable, occurring in old persons and depending upon organic changes in the central nervous system; the other, in younger persons, curable and probably not dependent upon organic changes. It is evident how untenable such a differentiation is, and that only the cases of the first class can properly be called paralysis agitans. The cases quoted by Jones from Reynolds, Graves, Sanders, and his own observations afford abundant proof of the superficial and uncritical manner in which the most varied conditions—of chorea infantilis, simple tremor without paralysis, etc.—were all grouped together under that curable form of paralysis agitans.

Anatomical Changes and Theory of the Disease.

The number of autopsies heretofore reported is not very great, and many of them, besides, give in part contradictory results, while others are entirely negative.

The oldest reports of positive changes are those by Parkinson and Marshall Hall; the former found in one case hardening of the pons, the medulla oblongata, and the cervical cord; Marshall Hall, a sclerosis of the pons and the corpora quadrigemina.—Stoffella found in a man, seventy-nine years old, in Oppolzer's clinic, who had been affected for five years, atrophy of the brain with secondary effusion of serum in the ventricles and the membranes, and an apoplectic cyst, the size of a pea, in the right optic thalamus; the pons and medulla oblongata were firmly indurated; the arteries at the base calcareous, and the lateral columns of the cord, especially in the lumbar region, permeated with grayish opaque lines, which, as well as the induration in the pons and medulla oblongata, consisted of new connective tissue.—Lebert mentions that in one case he found a shrunken sclerotic nucleus in the upper part of the cord, probably the result of previous inflammation.[1] —Cohn found in one case (a man forty-nine years old) well-marked cerebral atrophy, and in another (a man seventy-four years old), atrophy of the spinal cord at the level of the second cervical vertebra.—In a woman, thirty-four years old, who had been sick two years, and finally died from variola, Skoda found extended sclerosis of the central organs; the walls of the ventricles, fornix, pons, medulla oblongata and spinal cord were very firm; both optic nerves were flattened and sclerotic. In some opaque reddish patches of the brain the nervous tissue had disappeared, and its place was taken by newly-formed connective tissue, which likewise caused the sclerosis of the pons and medulla oblongata. There was besides œdema of the pia; the neurilemma of the nerves of the upper extremities was thickened; the muscles had undergone fatty degeneration. — M. Rosenthal found softening of the pons, of a portion of the medulla oblongata, and, accidentally, absence of the corpus callosum; Leyden, in paralysis agitans of the right side, found a sarcoma of the left optic thalamus and great flattening of the pons; Chvostek, affection of the left side, a hardening of the cortex of the right sphenoidal lobe and of the right hippocampus major, as a consequence of encephalitis; the large pyramidal ganglion cells of the hippocampus had completely disappeared, and only the neuroglia with the unchanged vessels remained.

Cayley, in a patient seen by Murchison (who died of typhoid fever after suffering twelve years), found changes in the spinal cord which, in part, depended upon chronic processes, in part, upon acute, which might well be referred to the typhoid fever. The former consisted of thickening of the cortical layer of connective tissue with increase of its nuclei; development of irregular tracts and islets of connective tissue, rich in nuclei, which penetrated from the cortex into the substance of the spinal cord, but only in the cervical and dorsal regions quite near the exit of the posterior nerve roots; the widely dilated central canal was filled with cells of different forms and sizes, some like leucocytes, some elongated, never having the character of normal epithelium.

In a case reported by Bourillon there seemed to be, judging from the imperfect description, a sclerosis in patches.

In the often quoted, symptomatically complicated case of Meschede, on section the ependyma of the lateral ventricle seemed covered with granulations with a spotted, marbled appearance. In the posterior lobe of the right half of the cerebrum were gray masses, which were in part connected with the cortex of the convolutions, in part were isolated. The medullary mass of the frontal and occipital lobes was markedly sclerotic, also certain parts of the optic thalami and corpora striata. The medullary substance of the cerebellum was as firm as cartilage, and pervaded by numerous gray islets; its corpora dentata were quite atrophied. Medulla oblongata and pons also atrophied. The spinal cord atrophied and hardened; in its middle portion, especially on the left, were small regions changed into gray masses. Microscopic examination showed, in the foci of the posterior lobe, ganglion cells and connective tissue elements; all the other abnormally gray parts showed the ordinary changes of gray degeneration.—In connection with these changes it seems to me that a statement made by Meynert[1] deserves notice, though not specially pertinent here. In a general paralytic, with a tumor chiefly on the right, Meynert found the weight of the corpus striatum and lenticular nucleus, including the isle of Reil, left side = 38 grms., right side = 44 grms.; the weights of the two frontal lobes were equal.

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[1] Handbuch der praktischen Medicin. Band II. 4. Aufl. 1871. S. 633.

Opposed to these positive results, Petraeus found (in the Copenhagen hospital) no changes in the nervous system; as apparent cause of death a fatty degeneration of the heart and pneumonic induration of the right lung. Also an autopsy by Ollivier furnished negative results. In a case described by Kuehne, from Frerichs' clinic (a man forty-nine years old, sick for five years, death from pleurisy and bedsores), there was nothing abnormal except œdema of the brain and spinal cord; the most careful microscopic examination of the pons and medulla oblongata, etc., showed nothing pathological.—Th. Simon (according to oral communication) found no changes in the nervous system nor in the sympathetic in four autopsies at the Hamburg hospital.—The detailed reports by Joffroy, the results of which were essentially negative, are of especial interest. They relate to four cases noticed by Charcot, all women (three of these are described in Ordenstein's dissertation). Macroscopically there was no change in the medulla oblongata and the spinal cord. The microscopic examination showed in three cases an obliteration of the central canal (by increase of the epithelium of the ependyma), a more or less well marked pigmentation of the ganglion cells, especially in Clarke's columns, and amyloid bodies in varying number. Besides, one case showed still the remains of an old circumscribed meningitis at the apex of the calamus scriptorius, and another a small superficial sclerotic spot in the medulla oblongata. The latter changes, on account of their inconstancy, must be looked upon as without significance as causes of the disease; but Joffroy also believes the changes, heretofore found to be constant, are to be considered as common phenomena occurring in all old people in consequence of senile degeneration. At most, according to his view, they can be considered as standing in a certain relation to senile tremor and the analogous trembling of paralysis agitans. According to this the origin of the tremor should be referred to the spinal cord. In the fourth case Joffroy missed also the above-mentioned changes, so that the result was entirely negative.—According to his latest statement, Charcot has made in all six autopsies in paralysis agitans; of these three were entirely negative, three gave results already mentioned.

If we look over the above-mentioned autopsies, we find cases in which there were absolutely no changes in the nervous system, or these were very slight, probably senile changes in the spinal cord (Petraeus, Ollivier, Th. Simon, Kuehne, Joffroy, Charcot); others showed exclusively changes in the spinal cord (Lebert, Cohn, Cayley, Murchison), or finally in the brain (Marshall Hall, Cohn, M. Rosenthal, Leyden, Chvostek); yet others, finally, showed extensive changes in the brain and spinal cord in the form of sclerosis (Parkinson, Stofella-Oppolzer, Skoda, Meschede). In view of such contradictions, we are at present compelled to be very cautious in forming conclusions as to the pathological anatomy of paralysis agitans and the theory of the affection. Not even can the question, whether the disease is of cerebral or spinal origin, be answered with certainty from the material already possessed, though, as we have seen, some facts at least seem to show that the origin of one initial and important symptom, the tremor, is in the spinal cord. Some authors (as Remak) have in a measure cut the knot by supposing a cerebral and a spinal form of the paralysis; the former (according to Remak) should be characterized symptomatically by a tendency to fall, while in the spinal variety this symptom is wanting. This distinction is, however, entirely artificial, and the case of the pretended paralysis agitans cerebralis, mentioned by Remak,[1] cannot with certainty be considered as paralysis agitans on account of the entire want of paralytic phenomena.

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[1] Wiener med. Presse. 1871. No. 25.

From the changes found in the brain must be excluded, as without significance, those depending upon senile atrophy (Cohn, Stofella), since undoubtedly they may also occur without the tremor. In other cases without senile atrophy or in addition thereto there are circumscribed foci of disease, sometimes in the optic thalamus (Leyden), sometimes in the hippocampus major (Chvostek), or in the pons and medulla oblongata (Marshall Hall, Rosenthal, Stofella). The localities of these foci are so various that we are prevented from forming any conclusions in regard to the pathogenesis of paralysis agitans. Certain symptoms (*e. g.*, the glycosuria noticed by Topinard) as also certain autopsies favor the supposition entertained by some authors, that the pons and upper part of the medulla oblongata must be considered the original seat of the disease; on the other hand, the same supposition is in decided opposition to the autopsies where that part of the brain has been found normal, and to the case of Larcher who, in isolated sclerosis of the pons, found symptoms which differed entirely from those of paralysis agitans.—Certain cases, finally, in which were found extensive changes of the central nervous system, although the symptoms are more or less similar to those of paralysis agitans, must probably be regarded as multiple sclerosis in patches (Parkinson, Skoda).

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[1] Galvanotherapie. p. 447.

Diagnosis, Prognosis and Treatment.

The diagnosis of paralysis agitans may, especially in the commencement, be difficult. It is most easily confounded with simple, senile or toxic tremor, and with disseminated sclerosis of the nerve-centres. Paralysis agitans may, however, be distinguished from senile trembling by the greater intensity of the tremor, by its occurring before senescence, in its further course more certainly by the addition of paralysis and the other disturbances of innervation. The history of the case is especially valuable in distinguishing tremor mercurialis, saturninus, alcoholicus, etc. The statements in the previous chapter furnish proof also how the clinical history, especially of tremor saturninus and alcoholicus, deviates essentially from that of paralysis agitans, while, however, tremor mercurialis very closely resembles it. Close attention will guard against mistaking between this disease and the rare affection lately described by Hammond as "athetosis," and the same is true in regard to chorea.

The differential diagnosis between paralysis agitans and sclerosis of the nerve-centres disseminated in patches is of especial importance. In this, too, there is usually trembling and progressive paralysis; there may be further disturbance of speech, contractions, paraplegia, and partial anæsthesia, more rarely even mental disturbance. The course in both affections is of many years' duration, and the prognosis is equally unfavorable. But in sclerosis the initial symptom is a progressive motor

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weakness, which increases to paralysis, while in paralysis agitans the tremor always precedes by a longer or shorter time; and also in sclerosis the paralysis begins, without exception, in the lower limbs, while the symptoms of paralysis agitans are first noticed in the upper extremities. The tremor in sclerosis is very much like that of paralysis agitans, but differs from it in that it is not spontaneous, only occurs during passive motions or when an active motion is contemplated. Less important differences are that in sclerosis sometimes headache and vertigo precede by some time the other symptoms; that irregular apoplectiform and cataleptic attacks often interrupt the course of the disease; that the reflex irritability is considerably increased, especially if the spinal cord is likewise affected with sclerosis; and finally, that sclerosis not rarely occurs in younger persons, between twenty and forty years of age.

The *prognosis* of paralysis agitans, as I remarked when speaking of the course of the disease, is absolutely unfavorable. The cases in which improvement or recovery have occurred must be considered as of doubtful diagnosis (see below). In individual cases the prognosis is so much the more unfavorable, that is, the fatal termination is to be expected so much the sooner, the earlier the paralysis and the other disturbances of innervation are added to the tremor, as also the older and the more debilitated the patient already is.

Under such circumstances the *treatment* can be almost only palliative. The best that can be hoped is that the disease should remain a long time stationary, or that single, especially tormenting, symptoms should be mitigated, as the tremor. Yet such a result can only rarely be attained.

Certainly in medical literature cases of recovery are reported, after, indeed, the use of very different modes of treatment. Elliotson has obtained such by means of carbonate of iron, Brown-Séquard by chloride of barium, Reynolds by the use of a Pulvermacher's chain, Remak by the constant current, Jones by hyoscyamus, Villemin by bromide of potassium, Betz by warm baths. If the "cured" cases are more critically considered, the identity of some with paralysis agitans is decidedly doubtful; in some the duration of the good result is not at all assured, but at most a temporary effect—disappearance for a while of the muscular tremor—is proven.

The case of Reynolds, for example, must have been quite recent, as the symptoms first appeared fourteen days before the treatment. There was vertigo and great numbness with trembling of the whole right upper extremity, the temperature of which in the region of the biceps was increased 4° F. An hour's use, five times repeated, of Pulvermacher's chain removed the tremor; continued treatment restored the power of the arm perfectly in one month!—In the (as to diagnosis very doubtful) case of Jones at first strychnia, iron, and ether, as also faradization of the arm were useless, even injurious, while hyoscyamus caused an improvement not otherwise characterized. Jones naïvely explains the inefficiency of the first agents and the efficacy of the hyoscyamus, by the statement that in his case, "as generally in chorea and epilepsy," there was increased irritability of the nerve-centres; hence, they required not the tonic but the calming medicines. Charcot has also obtained a quieting effect upon his patients by hyoscyamus, but it was only temporary. I have myself not been able to get any benefit from the long-continued use of the extract of hyoscyamus, or the hyoscyamin (advised by Oulmont for tremor).

Remak's case of pretended paralysis agitans cerebralis, already mentioned, must be considered at least of doubtful diagnosis, since no mention is made of paralytic symptoms. There was tremor, most marked on the left side, in the form of severe shaking and twitching of the limbs with tendency to fall forward, pain in the right side of the forehead and right temporal region, also conjunctivitis of the right eye. Remak supposed a stasis in the anterior lobe of the right cerebral hemisphere, and applied a stabile current to the temporal region. After eight days' treatment the pain in the temple and the eye disappeared, as also the tendency to fall and the tremor of the right side; the shaking of the left side remained unchanged.

Hitherto I have seen no good results from the use of the constant current (galvanization of the head and the sympathetics). Not once could any palliative effect be obtained by this means; it was not possible with the strongest current I could use to obtain even a remission or a temporary interruption of the tremor, as I had hoped. In one case I could use a current from fifty Siemens' elements passed through the head (the two electrodes placed on the mastoid processes), without causing vertigo, etc.;[1] even here there was no diminution of the tremor. The peripheral faradization and galvanization is likewise without effect.—Benedict also states that hitherto a cure of paralysis agitans by the use of electricity has not been obtained, though he thinks that the prognosis of this disease would be more favorable under early galvanic treatment. He has himself noticed a few good results in cases just beginning, and, besides, in one case a momentary effect upon the accompanying dyspeptic symptoms, for after galvanization of the sympathetic and the cervical enlargement there was immediately an improved appetite.

In Villemin's case, in which iodide of potassium (up to three grammes a day) was used with benefit, the diagnosis was not certain; probably, judging by the symptoms described, it was a case of multiple sclerosis.

The case described by Betz was that of a gardener, sixty-five years old, whose disease began half a year previously from the effect of catching cold, and was cured by six warm baths (27° R. [93° F.])!—I will take this opportunity to say that in genuine paralysis agitans the water cure is usually contraindicated. Especially objectionable are hot baths; lukewarm baths and those moderately cool are at least not injurious. Lebert thinks that by sea-bathing he has in one case caused the disease to remain stationary.

Concerning the treatment by corrosive sublimate, advised by Jones (but without personal experience), and that by strychnia, advised by Trousseau, I have no experience. According to Charcot, the latter agent seems rather to increase the tremor; Charcot also saw no favorable result from the use of ergotin and belladonna, which were used on account of their anti-spasmodic effect.

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[1] In aged persons it is quite often possible to use an unusually strong current to the head—probably on account of the senile thickening of the walls of the skull and the obliteration of many osseous canals, which very much increases the resistance.

The means which I have used in hopes of at least a palliative effect are: subcutaneous injections of morphia, of curare, and of Fowler's solution; also internal use of chloral hydrate, calabar, bromide of potassium, and nitrate of silver. The injections of morphia and curare produce occasionally a temporary cessation of the tremor; the subcutaneous injection of arsenic is much more successful in this respect, used in the form and dose advised under Tremor. In one case of well-marked paralysis agitans fifteen injections, in another four, produced a very marked diminution of the spasm, which in the first patient lasted two months. I have noticed no marked effect from the above-mentioned internal remedies either upon individual symptoms or upon the general course of the disease. Also Althaus found hydrate of chloral, Ogle calabar, Charcot nitrate of silver, useless in paralysis agitans; the latter seemed even to increase the spasmodic action, which (as Charcot rightly remarks) is all the more remarkable, as in multiple sclerosis it produced a considerable improvement and diminished the intensity of the trembling.

Appendix.

ATHETOSIS.

Hammond, Treatise on diseases of the nervous system. New York, 1872.—Med. Times and Gaz. 16 Dec., 1871. p. 747.—*Th. Fischer*, Athetosis. Boston Med. and surg. Journ. 30 May, 1872.—*Clifford Allbutt*, Cases of athetosis. Med. Times and Gaz. 27 Jan., 1872.—*Currie Ritchie*, Note on a case of athetosis, ibid. 23 March, 1872.

In 1871 Hammond described under the name Athetosis (ἄϑετος) a combination of symptoms somewhat resembling paralysis agitans, the chief characteristic of which is a ceaseless motion of the fingers and toes, which does not permit them to remain in any position in which they are placed. According to the descriptions given by Hammond himself and some other American authors, the involuntary motions observed consisted in part of alternate flexion and extension of the fingers and toes, in part of complicated motor phenomena, which were at times more severe, did not cease during sleep, could, however, be controlled and limited for a short time by position and extraordinary effort of the will. The phenomena seem to have had partially the character of associated movements, as while the fingers moved the arm was hard and rigid—during motion of the toes the muscles of the calf were in tonic contraction. In several cases there was at the same time disturbances of sensation (pain, sense of numbness or falling asleep); there was also change of temperature in the affected limbs.—Most of the observations were made on persons affected with epilepsy or mental disease; only in one case was there no other cerebral symptom. With the exception of the latter case, the patients were all men in middle life (between thirty-three and fifty-five years). As to treatment, bromide of potassium proved of service in two cases; in one case a four months' use of galvanization and faradization gave no marked result.

No autopsies have as yet been made. Hammond supposes the seat of the affection is in the intracranial ganglia, or in the upper portion of the spinal cord. It is more likely that the combination of symptoms known as athetosis is dependent upon changes, possibly circumscribed, of the centres of motor innervation in the cerebral hemisphere. The characteristic limitation to certain groups of muscles, the frequent occurrence of associated movements in the form of contraction, the association with epileptic attacks, seem—according to the investigations of Hitzig—to support the supposition of an irritation of these centres. In this connection it is to be remarked that the athetosis may be unilateral. In Fischer's case it was limited to the fingers of the right hand. I have myself lately seen a case which may be considered as belonging here, where the left foot alone was affected. So soon as the patient—a man thirty-three years old—touched the floor lightly with the tip of his foot, there immediately began a rhythmical alternate contraction of the flexors and extensors of the toes, to which was added tonic contraction of the muscles of the calf, which ceased as soon as the whole sole of the foot was firmly planted on the floor. (In this case there were still other symptoms, which led me to give a diagnosis of disseminated sclerosis.)

CHOREA.

VON ZIEMSSEN.

CHOREA.

Chorea St. Viti. St. Vitus' Dance.

The older literature is very extensive, and is presented very completely in *Wicke's* monograph.—*Sydenham*, Schedul. monitor. de novæ febris ingressu. Opera univ. med. edid. *Kühn.* Leipzig, 1827. p. 468 ff. und p. 531.—*Wichmann*, Ideen zur Diagnostic. III. Ausg. Hannover, 1827. Bd. I.—*Ketterling*, Darstellung des Veitstanzes. Regensburg, 1805.—*Bouteille*, Traité de la chorée ou danse de St. Guy. Paris, 1810. — *Bernt*, Monographia Choreæ St. Viti. Prag, 1810. Deutsche Ausgabe mit Zusätzen von *Sohler*. Prag, 1826.—*Wagner*, Pathologisch-therapeutische Abhandlung über den Veitstanz. Würzburg, 1817.—*Rufz*, Recherches sur quelques points de l'histoire de la chorée chez les enfants. Archives générales, 1834, Févr. p. 215 ff.—*Roth*, Ueber den Veitstanz und einige ihm verwandte Formen. Dissertation. München, 1843.—*Wicke*, Versuch einer Monographie des grossen Veitstanzes und der unwillkürlichen Muskelbewegung. Leipzig, 1844.—*Sée*, De la chorée. Rapports du Rhumatisme et des maladies du cœur avec les affections nerveuses et convulsives. Mémoires de l'Académie nationale de Médecine. Vol. XV. Paris, 1850. p. 373 ff. — *Leudet*, Sur les chorées sans complicat. terminées par la mort. Archives générales. Sept., 1853. p. 285.—*Romberg*, Lehrbuch der Nervenkrankheiten. Bd. I. S. 510 ff. Berlin, 1853, und Klinische Ergebnisse. Berlin, 1846. S. 19 ff.—*R. B. Todd*, Clinical lectures on paralysis. II. Edit. London, 1856. p. 428 seq.; and Lectures on Chorea. Lancet, April, 1843.—*Moynier*, Sur le traitement de la chorée. Archives générales, July, 1854.—The same, Journal de Méd. de Bruxelles, 1858. No. 59.—*Mosler*, Chorea bei Schwangeren. Virchow's Archiv. Bd. XXIII. S. 149. 1862.—*Kirkes*, On chorea, its relation to valvular disease of the heart, and its treatment. Med. Times and Gazette, 1863. Nos. 677 and 678.—*Bricheteau*, Relation d'une épidémie de chorée, observée à l'Hôpital Necker. Archives générales, 1863. I. p. 433 seq. and 532 seq.—*Hillier*, Chorea in connection with heart disease. Med. Times and Gazette, 1863, August 8. p. 142.—*Fischl*, Chorea minor in gravida. Wiener allg. med. Zeitg. No. 16. 1865.—*Hine*, A case of myelitis, presenting all the symptoms of severe Chorea.

Death on ninth day. Autopsy. Med. Times and Gaz. 788. 1865.—*Cyon*, Die Chorea und ihr Zusammenhang mit Gelenk-Rheumatismus, Peri- u. Endocarditis. Medic. Jahrbücher. Zeitschrift der Gesellsch. der Wiener Aerzte. Bd. XXI. p. 115. 1865.—*Russel*, Med. Times and Gaz., 1866.—*Monckton*, A case of Chorea, ending fatally, complicated with disease of heart and brain. Brit. med. Journ. No. 305. 1866.—*Roger*, Recherches cliniques sur la chorée, sur le rhumatisme et sur les maladies du cœur des enfants. Arch. génér., 1866. II. 1867. I. 54–75.—Ibidem, 1868, Jan.—Avril. Gazette des Hôpitaux, 1870. Nos. 65 u. 66. —*Richter*, Sitzungsberichte der Dresdener Gesellschaft für Natur- und Heilkunde. 5. Jan., 1867.—*Tuckwell*, Some remarks on maniacal chorea and its probable connection with embolism. Brit. and For. Med. Chir. Journal. October, 1867.—*Sam. Wilks*, Med. Times and Gazette. p. 117. 1868.—The same, Brit. med. Journ. July 30. p. 115. 1870.—*J. W. Ogle*, Remarks on Chorea St. Viti. Brit. and For. Med. Chir. Review, Jan. 1868.—*Th. Meynert*, Ueber die geweblichen Veränderungen in den Centralorganen des Nervensystems bei einem Falle von Chorea minor. Allg. Wiener med. Zeitung. Nos. 8 u. 9. 1868.—*R. Arndt*, Chorea und Psychose. Archiv f. Psychiatrie und Nervenkrankheiten. Bd. I. 1868.—*Steiner*, Klinische Erfahrungen über Chorea minor. Prager Vierteljahrsschrift. 1868. IV. S. 45.—*The same*, Zum Wesen der Chorea major und der Katalepsie. Jahrb. f. Kinderheilkunde. N. F. II. S. 205 ff. 1869.—*The same*, Epidemie von Chorea minor. Jahrb. f. Kinderheilkunde. N. F. III. S. 291. 1870. —*Kretschmer*, Ueber den Veitstanz. (Frerichs) Dissertation. Berlin, 1868.— *J. Hughlings Jackson*, Observations on the physiology and pathology of hemichorea. Edinb. Med. Journ., October, 1868. p. 294 ff.—Med. Times and Gazette, March 6, 1869.—*Douglas*, On the use of Indian Hemp in Chorea. Edinb. Med. Journ., March 1869. p. 777 ff.—*Barnes*, Chorea in pregnancy. Medic. Times and Gazette, 1868. Aug. 1; and Obstetrical transactions X. p. 147. 1869.—*Lawson Tait*, Dublin Quarterly Journal, Febr. 1869, p. 203. — *Broadbent*, Remarks on the pathology of chorea. British Med. Journal. April 17, 24, 1869.— *Carville*, Gaz. méd. de Paris. No. 50. 1869.—*Tuckwell*, St. Barthol. Hosp. Rep. V. p. 86. 1869.—*Mohamed Said*, De la Chorée et de la douleur, provoquée chez les choréiques. Thèse. Paris, 1869.—*Leidesdorf*, Ueber Chorea minor in ihrer Beziehung zu psychischen Störungen. Wochenblatt der Ges. der Wiener Aerzte. No. 12 u. 13. 1869.—*Fried. Weber* (Chor. gravid. 1 Fall). Berliner Wochenschrift VII. 5. 1870.—*J. Russel*, Med. Times and Gazette. 1868, May 30; 1869, Jan. 16; 1870, Jan. 8; April 2.—*Frick*, Ueber Chorea. Dissertation (Frerichs). Berlin, 1870.—*Legros* und *Onimus*, Recherches sur les mouvements choréiformes du chien. Comptes rendus, 1870. LXX. p. 1046 et seq.—The same, Quelques recherches sur les mouvements, Journal de l'Anatomie et de Physiologie. 1870. No. 4.—*E. L. Fox*, Med. Times and Gaz. p. 423. 1870.—*Gray*, Ibid. Dec. 17, 1870. — *Clouston*, Journal of Mental Science. 1870.—*Sieckel*, Chorea gravidarum. Diss. inaug. Leipzig, 1870.— *Gray* and *Tuckwell*, Cases of Chorea, treated on the expectant plan. Lancet. Dec. 2 and 9, 1871.—*L. Meyer*, Chorea und Manie. Archiv für Psychiatrie

u. Nervenkrankheiten. II. S. 535 ff. 1871.—*Dickinson*, Clin. Lectures on Chorea. Lancet I. 15. 1871.—*H. T. Butlin*, Cases of Chorea, treated with Sulphate of Zinc. Lancet, 1872. Nos. 17 u. 18.—*Ritter*, Gedanken über die anatom. Grundlagen der Chorea. Memorabilien. 1872. No. 10.—*G. Huntington*, On chorea. Philadelph. Med. and Surg. Reporter. 1872. p. 15.—*C. H. Jones*, Clinical lecture on cases of chorea. Med. Press and Circul. Jan. 10. p. 25. 1872. —*Fehling*, Chorea gravid. Archiv für Gynäkologie. VI. 1. S. 137. 1873.—*R. Arnoldi*, Ueber Chorea gravidarum. Diss. inaug. Berlin, 1873.—*A. W. Foot*, Unilateral chorea, disease of the corpus striatum and optic thalamus on the side opposite to affected. The Dublin Journ. of Med. Science. October, 1873.—*E. Long Fox* (Congenital Chorea), The British Med. Journ. 1873. No. 653.—*Landouzy*, Gazette méd. de Paris. No. 24. 1873.—*W. Leube*, 3 in ätiolog. Connex zu einander stehende Erkrankungen an Chorea St. Viti. Klinische Beilage zu den Correspondenzblättern d. Allg. ärztlichen Vereins von Thüringen. 1874. No. 5.—*De Beauvais*, Deux observations de chorée mortelle. Gaz. des Hôpit. 1874. Nos. 150 u. 151.—*P. H. Pye-Smith*, Guy's Hospital Reports. 3. Ser. XIX. p. 341. 1874.—*Weir Mitchell*, Post-paralytic chorea. The Americ. Journ. of the Med. Sciences. 1874. Vol. 136. p. 352 et seq.—*C. Golgi*, Sulle alterazioni degli organi centrali nervosi in un caso di Corea gesticolatoria associata ad alienazione mentale. Rivista clinica. 2. S. IV. 12. p. 361 (Reprint). Bologna, 1874.—*M. Benedikt*, Nervenpathologie und Elektrotherapie. II. Aufl. I. Abth. S. 251 ff. Leipzig, 1874.—*Gotthold*, Ueber Chorea. Diss. inaug. Würzburg, 1874.—*Bodo Wenzel*, Ueber Chorea major und minor. Schmidt's Jahrbücher. Bd. 162. S. 193 ff. 1874.—*Weinberger*, Zur Pathogenese und Therapie der Chorea. Wiener med. Presse. XV. No. 47. 1874.—*Pollock*, Treatment of Chorea with Inhalations of Chloroform. Lancet I. 25. p. 871. 1874.—*Elischer*, Ueber die Veränderungen in den peripher. Nerven, im Rückenmark und im Gehirn bei Chorea minor. Virchow's Archiv. Bd. 61. 1874. S. 485 und Bd. 63. 1875. S. 104 ff.—*M. Rosenthal*, Klinik der Nervenkrankheiten. II. Aufl. S. 576 ff. Stuttgart, 1875.—*Charcot*, De l'hemichorée post-hémiplégique. Progrès méd. 1875. Nos. 4 and 6; and Gaz. des Hôp. 1875. No. 30.

History, Definition.

The origin of the term chorea (Χορεία, dance) or St. Vitus' dance, and of most of its synonyms: St. Modestus' dance, saltus Viti, St. John's dance, ballismus (βαλλισμός, dance), choreomania, orchestromania, epilepsia saltatoria, danse de St. Guy, etc., is traced to the middle ages; in their original application these phrases signified simply that pandemic dancing-mania, which made its first appearance as a wide-spread mental disorder after the disappearance of the plague in the second half of the fourteenth century, in the neighborhood of the Rhine.[1]

In the year 1375 the psychical pandemic disorder extended from Aix over the whole region of the Rhine and Moselle as far as Holland. In the records of the time it is still spoken of as St. John's dance. The name of St. Vitus' dance is first met with in the Strasburg epidemic of 1418, when, under the magistrate's order, those affected were led in troops to the chapel of St. Vitus in Zabern, that they might be quieted by processions, masses, etc.

As regards St. Vitus, the legend informs us that he removed from Sicily when a boy, at the time of Diocletian's persecution of the Christians in the year 303, and suffered martyrdom in Florence, in company with Crescentia and his tutor Modestus. His mortal remains, after repeated removals, are said to have been interred in the cloister of Korvey in 836. This cloister, and other churches and chapels, erected to his memory, became subsequently objects of pilgrimage for sick persons of all sorts, and especially for those affected with the plague of dancing.[2]

As a pandemic disease, the dancing-mania died out in the fifteenth century, but traces have remained upon the Rhine till our own days, and especially in the so-called dancing procession ("spring procession") to the grave of St. Willibrod of Echternach, the origin of which can be traced to the plague of 1376, which passed through Luxemburg, and is contemporaneous with the great epidemic of dancing-mania in Aix and Cologne.

Subsequently the name chorea St. Viti was extended to the sporadic cases of spasmodic movements of the body, those forms of disease which nowadays are usually called chorea magna and chorea minor. But the so-called tarantismus and the raphania were finally included under chorea.

The confusion which arose from this throwing together of most unlike processes rendered it impossible to form a definite idea of the separate forms of disease. This may be seen most clearly in the dreary chaos of the literature of chorea compiled by Wicke in his complete monograph upon the malady.

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[1] "At Strasburg, many hundred began
To dance and leap, both wife and man,
In open market, lane and street,
By day and night; many did not eat
Until to an end their madness came;
St. Vitus' dance they did it name."

—Königshoven, the oldest Strasburg chronicle. Edited by *Schiltern*. Strasburg, 1698. p. 1085. Quoted by *Hecker*, Die Tanzwuth. p. 7. (Epidemics of the Middle Ages.)

[2] Cf. *Hecker*, Die Tanzwuth; and *Haeser*, Die Geschichte der epidem. Krankheiten. Jena, 1865. p. 172 et seq.

It is undoubtedly Sydenham that first evolved the modern conception of chorea from the mass of foreign matters that surrounded it in the middle of the seventeenth century. His description leaves nothing to be desired for clearness and precision:

> Chorea St. Viti convulsionis est species, quæ ut plurimum pueros puellasve a decimo aetatis anno ad pubertatem usque invadit; primo se prodit claudicatione quadam vel potius instabilitate alterutrius cruris, quod aeger post se trahit fatuorum more; postea in manu ejusdem lateris cernitur, quam hoc morbo adfectus vel pectori vel alii alicui parti adplicatam nullo pacto potest continere in eodem situ vel horæ momento, sed in alium situm aliumve locum convulsione quadam distorquebitur, quidquid aeger contra nitatur. Si vas aliquod potu repletum in manus porrigatur, antequam illud ad os possit educere, mille gesticulationes circulatorum instar exhibebit; cum enim poculum recta linea ori admovere nequeat, deducta a spasmo manu, huc illuc aliquamdiu versat, donec tandem forte fortuna, illud labiis propius adponens, liquorem derepente in os injicit atque avide haurit, tamquam misellus id tantum ageret, ut dedita opera spectantibus risum moveret (l. c. p. 468).

Sydenham essentially completed the separation of chorea from the foreign elements that had been confounded with it. Nevertheless, the idea has subsisted till quite lately, that there is some close relationship between the chorea minor sive Anglorum and the chorea major sive Germanorum, names used since Sydenham chiefly upon Wickman's authority. In the writers of the last century we find again the confusion of chorea with the pandemic diseases of imitation, with somnambulism, and even with raphania.[1]

The retention of the term chorea has helped to keep up the confusion of ideas. For this reason several attempts have been made to change the name for one more significant. Schaeffer,[2] who regarded the two so-called forms of chorea only as different grades of development of the same disease, proposed, towards the close of the last century, to give to our chorea the designation of

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[1] Cf. *Hofmeyer*, Von der Grübel- und Krumen- oder Schwerenothskrankheit. Berlin, 1742.—*Brendel*, Praelectiones academicae. Bd. II. p. 86 seq. 1793.—*Bouteille*, Traité de la chorée. Paris, 1809.—*Bernt* (l. c.).

[2] Salzburger med. chir. Zeitung. 1793. IV. S. 248.

"morbid irritability or muscular unrest;" and Thilenius,[1] in the same spirit, proposed the name "involuntary muscular motion." Wicke adopted the latter definitively, "in order to get rid of the notion that both diseases were but different forms of one species of disease."

These propositions have not been received with favor. Contrary though it be to common sense to call the grotesque performances of folie musculaire a dance, yet the term chorea has made itself so much at home that the attempt to supplant it by a fitter one could but fail. At the present time, while our knowledge of the anatomical disturbances which form the basis of chorea is in its infancy, we are simply unable to substitute a name based upon pathological anatomy for that taken from symptoms; so that the attempt to find a new name is hardly worth the while.

Yet, I must insist upon having the name of chorea restricted to the disease which is about to be described, and that *the so-called chorea magna sive Germanorum should be entirely ruled out.*

It is my conviction that *the group of symptoms called chorea major is not a disease sui generis, but is only the product of genuine psychoses and cerebral maladies* on the one hand, and of *hysteria and wilful simulation* on the other, such as so often and so abundantly flourishes in hysterical ground at the period of puberty. I have been led to this view both by a thorough study of the literature of the subject and by a series of very characteristic cases observed by myself; and neither Hasse's (l. c.) and Rosenthal's (l. c.) statements, both of whom regard chorea major as a distinct species of disease, nor the recently published observations of Franque,[2] Steiner,[3] Bohn,[4] Bodo Wenzel,[5] and Wysocki,[6] have been able to convince me of the contrary.

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[1] Med. chir. Bemerkungen. Th. II. S. 4 ff. Frankfurt a. M , 1814.

[2] Journal für Kinderheilkunde. 1867. S. 226.

[3] Jahrbuch f. Kinderheilkunde. N. F. Bd. II. S. 205. 1869.

[4] Ibid. S. 194. 1874.

[5] Schmidt's Jahrbücher u. s. w. Bd. CLXII. S. 195. 1874.

Wiener med. Wochenschrift. Bd. XXV. Nos. 14 and 15. 1875.

That Proteus, which is described under the name of chorea magna, contains in reality but one characteristic mark, and that is the associated spasmodic movements which are often performed with a certain fitness, but usually have an extravagant and violent character. But we have the same right to count all the associated spasms of hysterical patients as chorea major—the spasms of single extremities as well as those of the whole body, those of the muscles of respiration as well as those of the larynx. By the same right also all those striking forms of associated spasm which are observed in insanity, epilepsy, in cases of cerebral tumor, etc., must be added to the species "chorea major." I cannot regard it as justifiable to select a single group of symptoms from diseases of such varied character, solely on account of its striking nature and upon the authority of tradition.

As illustrations, let me offer a few instructive instances from my own practice.

Case I.—*So-called chorea major in an hysterical woman.*

M. B., a delicate young lady of eighteen years, with anteflexio uteri, decidedly hysterical, and to all appearances an onanist, over-indulged by her weak mother, was said to have suffered in the summer of 1873 with spasm of the glottis and a remarkably deep and hollow-sounding cough. Since the autumn of 1874 she has suffered from periodical spasms of respiration, with enormous acceleration of the rate of breathing (up to eighty in the minute), beginning usually in the forenoon and lasting four or five hours. Since Christmas she has suffered from associated spasms of the whole body, commencing towards evening at very nearly the same hour, and going on in the maddest way till near midnight. The patient performs like a complete acrobat; sometimes her body rolls itself into a ball, again it springs out to its full length, and again opisthotonus occurs, in which the body is bent far backward, is perfectly rigid, and its whole weight is supported by the skull and the tips of the feet. During this exhibition she utters the greatest variety of inarticulate sounds. The consciousness does not seem to be lost.

Rapid improvement in a cold-water establishment.

Case II.—*So-called chorea magna in an hysterical woman.*

S. D., æt. seventeen (in the medical policlinic of Greifswald), hysterical, daughter of an hysterical mother, suffers from violent spasms, of which she usually predicts the coming. Called to such an attack, I find the patient held upon the bed by four compassionate neighbors "to prevent her doing herself harm." She strikes about, foams, rattles in her throat, barks like a dog, etc. When let alone she creeps about the room on all fours, leaps on tables and cupboards, imitates the noise of various animals. Powerful induction currents and the pouring on of cold water stop the attack quickly and with equal success on each subsequent occasion, but they also have the effect of making the patient scold fiercely.

CASE III.—*So-called chorea magna in a case of cerebral tumor.*

H. W., æt. forty, a shepherd (at the 2d medical clinic in Munich), has suffered for years with the symptoms of a cerebral tumor (headache, dizziness, paralysis agitans of the right arm, etc.), and has presented the severest associate spasms for a long time. The latter occur quite regularly every third or fourth day, and last an hour or two. During these attacks the patient expends an enormous muscular force in performing the strangest involuntary movements (Zwangsbewegungen, compulsory movements), especially rolling of the body; continually repeated efforts to turn over backward, in which the head and neck strike the pillowed side of the bed with full force and the legs dangle in the air; the most rapid shaking of the head and right arm together; striking upon the chest with the fist; crossed jerking of the extremities; violent tonic spasms of breathing, etc.—In the course of the summer a mental affection, in the form of general confusion of ideas, supervenes. The patient declares that his clothes have been changed, is very irritable and fractious, runs away from the hospital in bare feet, wanders by night, etc.

CASE 4.—*So-called chorea magna, simulated.*

C. B., æt. fourteen (medical clinic of Erlangen), carpenter's apprentice to a strict master; after every trifling correction he is attacked by apparent unconsciousness, with general associated muscular spasms; he is removed to the house of his weak and credulous parents — and how often is the false indulgence of parents a cause of chorea magna! — where the attacks increase in violence, and assume the form of the so-called greater St. Vitus' dance. Of course, great sympathy is expressed for the boy; he becomes an object of universal curiosity, and at the request of his orthodox parents prayers are offered in the churches, he affirming that God alone can aid him. Finally, however, at the urgent instance of his relatives, in spite of much opposition from the boy and his parents, he is brought to the medical clinic. Here I had opportunity directly to observe the first attack. It was without question simulated, and was arrested instantly by the application of severe induced currents. The same occurred at the second attack. The patient, unmasked, makes his escape privately; the attacks have not returned.

CASE 5.—*So-called chorea magna, simulated.*

A. L., æt. twelve, son of a gardener. (Medical policlinic of Greifswald.) Great reluctance to attend school. Repeated punishments are followed by attacks of chorea magna, with the character of an impulse to run and climb, which are most distinct when there are spectators present. He mounts upon the stove, the wardrobes, climbs upon the trees, the roof of the house, and attracts great attention. He is recognized as a pretender, and is rapidly cured by cold affusions. He subsequently perpetrates many other impostures.

Hereditary tendency to hysteria, bad education, indulgent parents, credulous physicians, play the chief part in the production of these degenerate forms of hysteria, and in exaggerating the simulated spasms to the form of chorea magna. A characteristic feature, as it always seemed to me, was the surprising effect of severe, sudden, unexpected stimuli, like powerful electric currents or cold affusions. When once these means have been employed without regard to the patient's feelings, it is enough on a second occasion simply to bring the induction apparatus or the water-jug, in order to cut short the attack in the beginning. But these remedies have no effect, or but very little, in the general spasms of epilepsy or cerebral disease.

Cases will occur in which it is exceedingly hard to tell whether psychosis, cerebral disease, hysteria, or simulation is before us ; this is confirmed by the recently published observations of the above-cited authors.

Definition.

In the absence of a thorough comprehension of the nature of chorea, it is at the present time quite impossible to define the disease. We can only aim at a grouping, as complete as may be, of the most important points of this peculiar set of symptoms. But even this is attended with great difficulties, owing to the multiplicity of the phenomena ; and the separation of essential from non-essential must remain subject to the individual judgment of authors.

We understand by chorea a neurosis, of which the seat (as it seems) may sometimes be the brain alone, sometimes the entire nervous system ; characterized by incessant incoördinate twitchings or jerks of groups of muscles, which sometimes are spontaneous in origin, and sometimes are excited by voluntary impulse, which occur almost exclusively in the waking state, and are accompanied by a more or less developed psychical disturbance.

Etiology.

Chorea is mainly a disease of the time of bodily development; the period most affected is that from the second dentition to the close of puberty.

The statistics of the hospital for children at Paris are said by Sée (l. c., p. 448) to have included in twenty-two years 531 chorea patients. Of these there were 28 under six years of age, 218 from six to ten, 235 from ten to fifteen. A careful tabulation of the cases of which the beginning was known (191 in all) gave the following figures. The disease began

At less than 6 years	11 times.
From 6 to 11 "	94 "
" 11 " 15 "	57 "
" 15 " 21 "	17 "
" 21 " 60 "	12 "

The statistics of Rufz, Reeves, Steiner, etc., agree with the above.

Among Steiner's 52 patients four were less than six years old, 46 between six and eleven, and 6 between eleven and fourteen. In Guy's Hospital, Pye-Smith found

2– 5 years old,	5 patients	2 males,	3 females.
6–10 "	62 "	15 "	47 "
11–15 "	44 "	17 "	27 "
16–20 "	19 "	3 "	16 "
21–26 "	5 "	0 "	5 "
38 "	1 "	1 "	0 "

In the middle and later periods of life the disease is very rare, but distinct cases have been observed in the forties and fifties by Andral, Reeves, and Frank; and in the sixties and seventies by Jeffreys, Bouteille, Powel, and Maton. Next to these periods, that of infancy is the least exposed; and the number of cases grows less in proportion as we approach the period of birth (Sée). A few cases of chorea have been observed in infants at the breast by Simon and Constant, and even just after birth by Monod, Fox, and Richter.

Fox calls his case one of *congenital* chorea. The child, a male, was born six weeks before the period, and from the first hours of his life he suffered from violent choreic movements. In his third year he is said to have had epileptic attacks, but they did not occur either before or after that time. In his thirteenth year, when observed by Fox, the power of speaking and walking was defective, but intelligence was normally developed. The chorea was much improved by an arsenical treatment.

Richter also mentions congenital chorea in two girls, whose mothers had suffered a severe fright when advanced in pregnancy. The children suffered from their birth with clonic spasms which ceased during sleep, and at a later period almost wholly disappeared.

Of the sexes, the female is decidedly more troubled than the male. Of Sée's 531 patients, 393 were females, and 138 males. Among Rufz's patients in the children's hospital at Paris, the proportion of females to males was 138 : 51 ; with Hughes, 73 : 27 ; with Steiner, 40 : 12 ; with Pye-Smith, 106 : 42. In 27 adult cases, between the ages of twenty and seventy, Hasse found nearly the same proportion, viz., 19 : 8.

Hereditary transmission does occur, but is certainly rare. But hereditary *tendency* to chorea, as to other diseases of the nervous system—as hysteria, epilepsy, etc.—or rather, an hereditary transmission of a special susceptibility to irritation, an impressionability of the nervous system, on the one hand, and of a general weakness of the entire constitution on the other hand, is very often demonstrable.

untington, of Long Island, with his father and grandfather (also physicians), says he has observed entire families of choreic persons, in which the disease was propagated until once a generation had been overleapt, when the hereditary disposition in that family ceased. In these cases the chorea began between the twentieth and the fortieth year of life, attacked men and women alike, and usually led to mental disease, often associated with suicidal tendencies, and finally to death (?).

All circumstances which are adapted to increase the excitability of the nervous system at the period of *sexual development* are of great influence ; such as a bad, indulgent education, premature excitement of the sexual passion, onanism, psychical excitement, etc.

Fautrel[1] and Wendt[2] speak especially of the consequences of self-abuse, practised in great excess previous to puberty. Fautrel's patients were all onanists. It is impossible to get statistics regarding this point, so that it must remain hard to estimate its importance as a cause or a predisponent.

Among adults, we have to mention as predisposing circumstances *pregnancy*, and disturbances of *menstruation* and *sanguification* (chlorosis, anæmia).

The *immediate causes* may be very various. Psychical disturbances, whether acting acutely in the form of fright[3] or dread, or bringing to bear the slow depressing influences of sorrow and fear, of pain or discontent, are without doubt powerful agents in bringing the disease to an outbreak, especially in predisposed persons.

Among the mental causes must be included *imitation*, or what may be called the contagiousness of chorea. A number of accredited facts exist which prove the possibility of a propagation of the disease by psychical contagion among those children in the neighborhood who are predisposed through hysteria, nervous tendency, or education, as in the brothers and sisters of a patient, or the children of a boarding-school. Besides the older observations of Mullin,[4] Crampton,[5] Eckstein[6] and Uwins,[7] we must here mention the recent observations of Bricheteau and Leube.

Leube saw two girls, of the ages of sixteen and seventeen, hysterically predisposed, who acquired chorea in consequence of intercourse with another girl, aged twelve, who had the disease; but the trouble was of short duration, yielding quickly to galvanization and psychical treatment.

Bricheteau's observation is still more striking. Into the hôpital Necker, in a ward where there were girls who were hysterical or who had formerly been chorea-tic, a girl with chorea was admitted, and within six days eight other patients in the same ward were taken with chorea. The further spread of the disease was arrested by isolating the chorea patients. The intensity of the symptoms varied greatly; some of the attacks lasted for months.

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[1] *Sédillot's* Journ. génér. de méd., etc. T. XXXIX. p. 319. 1810.

[2] Kinderkrankheiten, Breslau, 1835.

[3] See, for instance, the case by *Romberg* (Nervenkrankheiten. p. 516) of a girl aged ten, who was severely frightened in the morning by a dog that sprang at her and barked loudly, and in the evening was seized by chorea. See also the observation on p. 432.

[4] Edinb. Med. and Surg. Journ. V. 1. 1805.

[5] Transactions of the College of Physicians in Ireland. V. p. 110. 1824.

[6] *Horn's* Archiv f. med. Erfahrung. Bd. III. S. 301. 1803.

[7] Edinb. Med. and Surg. Journ. V. p. 405. 1812.

Here we have to do with serious and protracted affections, the origin of which, in the way of contagion, can hardly be doubted. At all events, a *distinct predisposition* seems to be necessary to propagate the disease by imitation; otherwise the number of instances would be much greater than it is.

As regards the *geographic distribution* of chorea, the statements of Rufz (l. c.), that the disease is less frequent in the warm zones, has not been confirmed by the researches of Hirsch.[1] The question of the frequency of chorea in different countries, and of its extension to north and south, to east and west, is one which cannot at the present time be decided, even in a temporary way, for, as Hirsch observes, the statistical material of civilized countries comes only from hospitals and from the practice of single physicians, and therefore is valueless for scientific purposes; while the reports as to the existence of the disease in different parts of the world are so few that no general conclusions can be attempted at present.

The same is the case with the influence of *seasons* and *weather;* we have no useful statistical basis.

As regards the causal relation existing between chorea and *other diseases*, the case of *rheumatism*, and especially of *acute articular rheumatism* and its complications, deserves especial attention. From the beginning of our century the relation of chorea to rheumatism has been observed, and the English and French authors have taken special pains to show that articular rheumatism and its cardiac complications are an important source of chorea.

In judging of this question, we have scarcely any but French and English statistical reports at our disposal. The first statistics of the sort originated with Hughes, and were printed in Guy's Hospital Reports for 1846. They include 108 cases of chorea, of which 14 were complicated with rheumatism and heart disease.

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[1] Historisch-geograph. Pathologie. Bd II. S. 570. Erlangen, 1864.

Ten years later, the same author, in connection with E. Burton Brown, published in Guy's Hospital Reports for 1856 a second report, embracing 209 cases. Among 104, in which it was possible to make careful examination as to the history and the bodily condition as regards rheumatism and affections of the heart, there were but 15 in which rheumatic accidents had *not* preceded the attack, and a cardiac murmur could *not* be demonstrated. According to Kirkes,[1] almost all cases of chorea are caused by endocarditis.

Of the French authors, Sée and Roger are the most zealous upholders of the causal relation of chorea and rheumatic affections in the widest sense. Sée (l. c., p. 414) found among 128 chorea patients 61 who suffered at the same time with articular inflammations and pains in the joints.

This coincidence seems the more remarkable, as rheumatism is rare in childhood. Sée gives the following striking figures to show this. Among 11,500 patients received in the course of four years in the children's hospital there were only 48 cases of rheumatism without chorea, and 61 of rheumatism with chorea.

Roger finds the coexistence of the two so frequent, that he is inclined to regard it as the expression of a pathological law, and to regard both as one and the same affection under two forms.

Spontaneous acute rheumatism is far more common in children (according to Roger) than is usually supposed; that is, from the fifth year and upward. Before the fifth year it is certainly rare. As a sequel of scarlatina, called out by exposure to cold, rheumatism of the joints is common, is usually confined to the neck and arm, is of slight severity, lasts but a short time, and is more rarely complicated with affections of the heart, etc. The spontaneous articular rheumatism of children is oftener subacute than acute, and attacks only a small number of joints; but the frequency of the complications with endocarditis and pericarditis is none the less great, and it is the light cases rather than the severe that are complicated with chorea, which prefers the period of the decline of the disease for its own attack. According to Roger, children who have passed through rheumatism are predisposed to chorea by that circumstance, and *vice versâ*, choreic children are in danger of being attacked sooner or later by rheumatism. Both diseases have the same tendency to relapse.

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[1] Med. Times and Gaz. 1863.

Very different from these statements of English and French authors are the results of the observations made by Steiner[1] in Prague. He saw, among 252 cases of chorea, only four which originated during the course of acute articular rheumatism. Steiner expresses his suspicion that this wide difference is due to local influences.

My own observations, though few, tend to confirm Roger's statement. Among twenty-one cases there were four in which the first attack, or a relapse of chorea, followed acute articular rheumatism, directly or after a considerable time.

If we add to these observations the considerable list reported by older and newer writers (Copland, Seahouse, Forgues, Bright, Babington, Hughes, Monckton, Hillier, Romberg, Trousseau, etc.), who establish a causal relation between chorea and rheumatism, we cannot doubt the closeness of the connection. How to explain it will be our object in a later portion of this paper.

As regards the relations of chorea to other diseases, it may be connected with a great variety of inflammatory and other processes in the central organs of the nervous system and their membranes, or may develop from scarlatina, bodily injuries, or disturbances of the uterine functions; but causes of these sorts are so rare that we may regard them as merely constituting the last impulse towards the outbreak of a disease for which preparations are already made.

The relation of chorea to *pregnancy* deserves special mention. To the interest felt by the gynæcologists in this point during the last ten or twenty years we owe a number of careful essays, among which the statistics of Robert Barnes[2] are especially valuable. Barnes's collection of fifty-eight cases—fifty-six in pregnant women and two in women in child-bed—has been enlarged by Bodo Wenzel[3] by eight more cases, of which four come from

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[1] Prager Vierteljahrschrift, 1868. Bd. III. S. 51.

[2] Obstetrical Transactions. X. p. 147. 1869.

[3] Schmidt's Jahrbb. Bd. CLXII. S. 199 ff. 1874.

Sieckel,[1] one from Friedr. Weber,[2] one from James Russell,[3] one from H. Thompson and Hall Davis,[4] and one from Fehling.[5] According to these figures, chorea occurs oftener in primiparæ than in pluriparæ; of 51, 31 were primiparæ. The majority of the patients were from twenty to twenty-three years of age. Chorea begins oftener in the first than in the second half of pregnancy; of 57 pregnant women, the beginning of whose attack is noted, 22 were seized in the first three months, 23 in the second three, and 12 in the last three. No regularity or order can be discovered in the relation of chorea to pregnancy, as Wenzel carefully shows. Chorea sometimes occurs in persons who have already suffered from it during childhood or puberty; and in the above list of sixty-six there are found fourteen who had had chorea previous to pregnancy; sometimes it attacks women in pregnancy for the first time; sometimes it occurs in first pregnancies and not in later ones; sometimes it repeats itself in every subsequent pregnancy, or it may not appear at all till a later pregnancy; sometimes it begins in the first months and disappears before delivery; sometimes it begins in the later months and ceases at delivery, or lasts beyond it, or even makes its first appearance at the time of confinement. The latter case seems to be the rarest, and exists in only two of the sixty-six.

As a proximate cause of the attack, violent fright is assigned in seven cases. Rheumatism and endocarditis are noted as preceding the attack in only seven cases.

The relations of chorea to *mental disease* will be spoken of later.

Pathology.

General Description.

The disease is very often ushered in by disturbances of the mental faculties and the general health, of longer or shorter duration ; these are sometimes wholly absent. It is particularly in the very acute cases, caused by fright, that the characteristic muscular twitchings form a first symptom.

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[1] Inaugural dissertation. Leipzig, 1870.

[2] Berlin. klin. Wochenschrift. VII. 5. 1870.

[3] Med. Times and Gazette. Jan. 8, 1870.

[4] Lancet. II. October 15, 1868

[5] Archiv f. Gynäkologie. VI. 1. S. 137. 1873.

When there are prodromal symptoms they consist of a striking change in the temper, a fretful, discontented, or apathetic behavior in children formerly cheerful and brisk, unusual changeableness of temper, sudden and causeless change from sadness to excessive mirth, or the reverse. At the same time the mental energies fail, the patients become inattentive, forgetful, absent, and their schoolmaster is apt to be the first to notice this in their awkward and silly behavior, and to blame them for it. There also occur hyperæsthesiæ and paræsthesiæ in the nerves of sense and sensation, weakness of the motor apparatus, disturbance of appetite and sleep.

Of the characteristic *choreic disturbances of motion*, the first indication is given by a general restlessness of body, which will not let the patient remain long in one position, and by a remarkable clumsiness in performing common simple actions. The patients work about in their seats, drop things they are holding, their writing becomes bad and illegible, their sewing useless, their piano-playing confused. At this stage the disease is commonly not recognized by the parents and teachers, who punish it as carelessness or neglect. As the trouble grows worse, twitchings of groups of muscles in the arms, shoulders, and face occur, which the patient at first tries to conceal by executing a voluntary and rational movement directly after. But these involuntary twitchings soon extend down over the whole apparatus of animal motion, and not only interfere with willed actions, but by the frequency of their spontaneous occurrence keep the body in continual unrest. In spite of his best efforts, and the powerful influence of his parents' blame, his comrades' ridicule and the presence of strangers, the child is unable to prevent the muscular contractions for any length of time. Often the volition is not even able to diminish the spontaneous jerking ; in fact, the muscular unrest is often aggravated by the effort.

The want of order with which the muscular jerks pass from one member to another, the tireless activity of the muscles in spite of the purposeless and useless nature of the acts, the lightning rapidity of the jerks, and the slow and careful way in which intended motions are performed, the spasmodic grimaces, alternating with periods of expressionless, almost idiotic, rest, all contribute to form a most characteristic picture, which it is scarcely possible to mistake. Sometimes one arm is bent or extended; sometimes the fingers are spread apart; the shoulder raised; the head jerked in one or another direction; the features are drawn into the greatest variety of contortions; the eyes rolled hither and thither; the mouth is sometimes opened, sometimes closed; the jaws are separated and brought together, sometimes so violently that teeth are broken off;[1] the tongue is extended and drawn back with the velocity of lightning, or is rolled aronnd in the mouth. The spasms of the trunk give rise to active locomotor movements of the whole body; the patient is thrown from his chair or out of bed. The legs are actively engaged; sometimes they scuff on the floor when the patient is sitting; sometimes they are suddenly drawn to the trunk, so that if the patient is walking he falls, or is turned or hurled to one side or another. Thus the spasmodic play of the muscles leaps about in the maddest way, sometimes affecting one side more than the other, sometimes both sides. At last every purposed movement is impossible; the patient can no longer eat without help, as he spills everything, drops his cup, and fails to reach his mouth with the fork or spoon; he can no longer dress himself, and the disturbance in the apparatus for phonation and articulation prevents connected speech.

The functions of the sphincters of the bladder and rectum remain unaffected, as do those of the automatic apparatus of the heart, of respiration, and of deglutition as far as the latter is involuntary.

When the patient is once asleep, the muscular action almost always ceases, but it is very hard to get to sleep; the rest is imperfect, and is disturbed by bad dreams. On awaking, the scene of tumult begins again.

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[1] In a case by Tuckwell that ended fatally, in a girl of ten, three of the lower cutting teeth were broken off by the spasms of the muscles of mastication; the exposed gum was by degrees worn to shreds by the upper teeth.

The terms "insanity of muscles," "folie musculaire," applied by Bellingham and Bouillaud to the confused play of the muscles, is very appropriate.

The mental depression with which the disease begins usually increases considerably during its course ; the patients are freakish, obstinate, selfish, violent to excess even towards their parents ; and with these changes there is often joined a failure in intellectual power, with incapacity to think, imperfection of judgment, weakness of memory—which is extremely disquieting to parents, especially in the educated classes.

The improvement in all the symptoms takes place in most cases very gradually, and is often interrupted by relapses. Psychical changes are especially unfavorable, and may greatly retard convalescence.

Analysis of the Symptoms.

Disturbances of the motor functions.—The involuntary muscular contractions, which are the characteristic feature of this disease, either occur spontaneously, or as the result of an attempt to perform some ordinary action, which provokes irregular associated movements. In light cases the latter are the more common, and the spontaneous jerkings are few. In severe cases the former are the more frequent; the whole body is continually thrown about by the explosive muscular action; the extremities are thrown out, etc. The disturbing associated acts which accompany intended motions are less noticed in such cases, because the patient, feeling himself incapable of every act, is as passive as possible. He must be clothed, fed, led about, or even carried by his attendants.

The extension of the affection to the animal muscular system varies very much according to the severity of the case. In slight cases it is confined chiefly to the head, shoulders, and upper extremities ; but in severe ones there is hardly a voluntary muscle in the whole body but is affected to some extent, excepting the sphincters of the bladder and rectum, which always remain unaffected, and the muscles of the ear, regarding which we have no positive statement. A considerable degree of chorea of the muscles of the pelvis and lower extremities makes walking impossible, even with support on both sides. Not only does the patient involuntarily take such long steps that he loses his balance, but one leg or the other bends while walking, or is drawn up toward the pelvis so that he falls down. The choreic motions of the legs become very distinct when the patient is led by an attendant.

In one of my patients, a boy of ten, son of a tailor from the country, the chorea was caused by a sudden fright from hearing a shot fired unexpectedly close to him while gathering hops. It reached a great height in a few hours; the boy could not walk without support, for the long steps he took turned his body around, now to the right, now to the left, and made him fall. When led by two assistants he took enormous strides, but while doing this both legs were suddenly thrown across each other, or drawn up so that the heels touched the nates, and the patient, held up by the arms by his attendants, dangled in the air. When in bed the movements continued in the same manner. Sometimes the legs were drawn up to the body, sometimes both arms were thrown violently above the head, then down again, crossed over the breast, etc.

Some authors say they have seen crossed choreic contractions in which the muscles of the *right* arm and *left* leg are affected, or the reverse; but the reported observations are not demonstrative.[1]

Restriction to one lateral half of the body, the so-called *hemichorea*, is much oftener observed. In it the unilateral character is not confined to the extremities, but extends to the face and the apparatus for articulation and swallowing. It is remarkable that the *left* side is oftener affected than the right, and in bilateral chorea the left side is often worse than the right.

Ewart,[2] in 1760, reported cases of this sort of unilateral, especially left-sided, chorea, which were followed by those of De Haën,[3] Gardane,[4] and Rufz.[5] The fact was afterwards contested by Dugès,[1] but was established by the statistics of Sée and others. According to Sée's observations, out of 154 cases in which the seat of the motions was carefully noted, the affection was confined to the left side, or was more marked there, in 97 cases. Of pure hemichorea, he states the ratio of left to right side as 37 : 27. Pye-Smith observed, among 150 cases, 33 confined to one side, of which 15 were on the right and 18 on the left. Russell observed, among 97 cases, 29 confined to one side, and of 54 that involved both, the disease began as unilateral in 43.

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[1] *Woeltge's* case (in his Observationes Med. Göttingen, 1783, p. 5, cited in *Wicke*, p. 279), which forms the basis of the statements commonly made concerning crossed chorea, is of a very doubtful and ambiguous character. So are the observations of *Albers* (Hufeland's Journ. f. prakt. Heilk. I. S. 152 ff., 1795, given in *Wicke*, p. 459. Anhang).

[2] De chorea St. Viti. Edinburgh, 1760. Quoted by *Sée*.

[3] Prælectiones in H. Boerhavii instit. pathol. Viennæ, 1780. Tom. III. p. 624 seq.

[4] *Gardane*, Observ. en faveur de la Méd. Electr. Paris, 1768. Quoted by *Wicke*.

[5] L. c. p., 224.

Exhaustion of the muscles chiefly involved might be expected to follow such incessant activity; but it is remarkable that this does not occur. The patients complain of nothing but a general weakness. The explanation of this absence of local exhaustion is probably to be found in the excessively short duration of the individual contractions and the constant change in the seat of the spasm.

Complete repose of the muscles occurs during *sleep* in the great majority of cases, but rare instances occur in which the spasms do not entirely cease in sleep. These, according to Cyon, are cases of reflex origin (chorea reflexoria), and appear "in the peri- and endocarditis of articular rheumatism, in disturbances of the female generative system, in cases of intestinal worms, and perhaps with some cutaneous diseases." But it must not be forgotten that while a reflex origin of chorea seems to be very frequent, the persistence of the spasms during sleep is a very rare phenomenon.

The intoxication of chloroform and the sleep of chloral arrest the spasms at once; whether this is true in all cases cannot yet be determined. In all cases in which I have caused chloroform to be inhaled to the point of producing narcosis, or have given chloral hydrate—and these were all severe cases—the muscular disturbance ceased at once and completely.

After awaking the movements are at first moderate, but increase with the first attempts at voluntary motion, as in rising or dressing, and soon reach their former violence.

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[1] Essai physiologico-pathologique. Tom. I. No. 23. Paris, 1823. Quoted by *Sée*.

Bernt (l. c., p. 24) states that certain *positions of the body* may cause the appearance or disappearance of the spasms; he supports this by two observations by Tulpius and Thirmaier, and makes two classes—chorea sedentaria (Tulpius' case of a hysterical female who is said to have been affected with chorea only when in a sitting posture), and chorea stataria (Thirmaier's case of a man who was always brought to his feet by the spasm, although they tried to fasten him to his bed or arm-chair). But both cases are very doubtful, and cannot well be included with our chorea.

As regards the reaction of muscles and nerves under the *electrical current* little is known. M. Rosenthal[1] found distinct increase of the electro- (farado-) muscular contractility in three cases of hemichorea which had come quite early under his observation ; and the galvanic test showed a high degree of excitability, which appeared both in the fact that weak currents produced contractions at cathodic closure (KaSZ),[2] or even tetanic contractions (KaS tetanus),—and also in the contractions produced at cathodic opening (KaOZ). Benedikt[3] also usually found, in galvanic examination, that all the tests exhibited a considerable increase of reaction, and, in particular, that the cathodic opening reaction (KaOZ) was produced by a current very little stronger than that required for cathodic closure reaction (KaSZ).

No law can be deduced from these scanty data at present. Not only do we require a much larger collection of facts to enable us to answer the question whether chorea is always accompanied by an increase of the farado- and galvano-muscular excitability, but the observations ought to inform us separately of the condition of electrical excitability at the beginning, during the course, and in the decline of the disease. Finally, it is necessary to test the excitability of the muscles and their motor nerves *separately*, giving special heed that the conditions requisite to scientific accuracy of observation are observed. Unfortunately, the estimation of the degree of electro-muscular contractility is still very much a matter of individual judgment. For the present we can do no more than to establish with care the minimum of excitability by working with as constant a source as possible (Siemens's elements) and Siemens's rheostat.

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[1] Klinik der Nervenkrankheiten. p. 581.

[2] For an explanation of these formulæ, see Vol. XI. of this Cyclopædia, pp. 271-274.

[3] Nervenpathologie und Elektrotherapie. p. 256.

The occurrence of *painful points* and *points painful upon pressure* in chorea has been examined by Triboulet, Rousse and Perigault, and Mohammed Said, who have shown that the nerve-trunks of the affected regions, and even the parts covering them, are sensitive in various degrees to pressure, and that in the severest cases the patients complain of spontaneous pain. In hemichorea the affected side, with few exceptions, is alone painful; but if the other side afterwards becomes involved, its nerves become painful also. The painfulness is in direct ratio to the violence of the choreic movements.

A participation in the choreic movements on the part of the vegetative muscular system is generally denied, or is regarded as very rare. And, in truth, the facts which favor this view are too ambiguous to permit of such an interpretation. In the case of such automatic actions as the respiration, in which the will is capable of influencing the rapidity and rhythm of the movement, irregularities are often observed in the act, whether of inspiration or expiration, which can only be interpreted as choreic. I have paid especial attention to this point, and have reached the following conclusion:

Impaired or lost power of speech depends not solely upon the choreic disturbance of the functions of the muscles of articulation, but may be due to chorea of the muscles of the abdomen and glottis. The implication of the abdominal muscles is best shown by the effect produced upon the act of expiration, which, especially when forcibly performed, assumes an irregular, jerking character. The effect is most marked when the patient is caused to sing, or to speak continuously as in counting. In slight cases this produces only a deficiency in the power of continuing the equal pressure of expiration required for vocalization; the pressure soon ceases, and rather suddenly, so that the patients can bring out only a few syllables or numbers, which are interrupted by a hasty inspiration. In severe cases they can utter only one syllable or number in one expiratory effort, and between every two syllables there is a remarkably hasty inspiration; others drive out their syllables or numbers forcibly, and their speech has something of an explosive character. As the case improves, the number of syllables which can be spoken with a single effort increases. The explosive contractions of the abdominal muscles are perceptible to the touch.

Chorea of the laryngeal muscles, of which no cases have been published, is in my observation a usual accompaniment of the severe form of the disease. It is characterized by insufficient force and duration of the tension of the vocal cords in phonation, owing to a want of co-ordination and persistence in the muscular act; the patient is unable to sustain a tone in singing for any considerable time, or to utter several words together without interruption. The singing tone is suddenly interrupted, and the last syllables of the words are cut off in the effort to make the next inspiration. In reading aloud a great deal of muscular force and of breath is expended, so that the patient is soon very tired. That the tension of the vocal cords in phonation is often insufficient I have been able to show in several cases by the relatively *deep pitch* and *monotony of the voice*, which disappeared as the patient improved, giving place to the normal pitch and modulation of the voice. With the laryngoscope it is very easy to watch the restlessness of the laryngeal muscles, the twitching contractions of the closers, openers, and tensors of the cords.

Many authors speak of a *chorea of the heart*, by which they understand palpitations or irregularities of rhythm, which continue while the disease is at its height, but neither before nor after.

Cases of *arhythmia* are certainly very rare, and there is hardly anything published about it. Romberg (Nervenkrankheiten, p. 214) could not discover any abnormal variation in the movements of the heart, in spite of continued observation. Hasse (l. c., p. 167), on the contrary, observed two patients whose pulse occasionally intermitted without any demonstrable disease of the heart.

Simple *accelerated action of the heart*, sometimes with a full hard pulse, sometimes with a soft and small pulse, is often mentioned, and has been repeatedly observed by us. According to

Stiebel,[1] the palpitations are especially prominent during the decline of the disease.

Reeves[2] has lately described chorea of the heart as a peculiar nervous affection of the heart, often connected with chlorosis and general chorea, but also occurring apart from them, in which there occur nightly attacks of palpitation and distress for breath, with free intervals; attacks which, therefore, have a great resemblance to those of angina pectoris. In this description we can find nothing characteristic, nothing which differs from the nervous palpitations which are so frequent in young patients; and we wish to state clearly our belief that it would be very improper to give the name of chorea of the heart to anomalies of this sort in the rhythm and rapidity of the heart, as Reeves has done. Such a name would only lead to fresh confusion. In our opinion, the name chorea should be restricted to those arhythmiæ and palpitations which develop during, and as a consequence of, actual chorea. The influence of the restless muscular action upon the aortic pressure and the work of the heart must necessarily be counted as a factor which may influence the pressure and rate in the same way as a choreic disturbance of innervation may.

The *pupils* are very commonly *dilated*—a fact which most authors have reported, and which I have observed in almost every case. The reaction under the influence of light is often very much diminished, as Hasse, M. Rosenthal, and others, have stated. I have repeatedly seen the dilated pupils react very sluggishly to the impression of light; and in one case, where the chorea was chiefly localized in the right half of the body, and the right leg was a little dragged in walking, the pupils were both dilated, but the right considerably more so than the left. Of course, a dilatation of the pupil is a very common and often seemingly causeless phenomenon with children, and it is not proper to assign it a causal relation to chorea unless the pupil has been found to be of normal size both before and after the disease—a precaution which, it would seem, has scarcely ever been taken. It would be desirable to observe also the behavior of the pupils in sleep and during the muscular rest procured by chloral hydrate and chloroform.

M. Rosenthal, who once saw the dilatation of the pupil disappear spontaneously after the termination of the chorea, was not able, even by direct electrical stimulation, to change the pupil during the continuance of the disease; he therefore regards the phenomenon as the expression of a spasm of the dilatator pupillæ muscle, caused by irritation of the centrum cilio-spinale—an assumption, however, which is quite hypothetical.

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[1] Klin. Beiträge zur Heilwissenschaft. Frankfurt a. M., 1823. p. 43–67.

[2] Heart diseases in Australia with observations on aneurism of the aorta. Melbourne, 1873. Schmidt's Jahrbb. Bd. CLXI. 1874. p. 131.

In regard to this, as to so many other points, we are completely in the dark.

The *reflex motions* appear to be executed normally in all cases.

Sensibility appears, according to the observations which we have, but rarely affected. In these rare cases there has been sometimes observed a diminution of cutaneous sensibility in one or both halves of the body (Charcot), and of the acuteness of the senses of hearing, smell, taste, and sight; sometimes a general hyperæsthesia and hyperalgesia of the skin and the senses. But the small number of the observations made upon this point, and their incompleteness, and especially the neglect to test the kinds of cutaneous sensibility separately, render it undesirable at the present time to make much use of them.

The feeling of *muscular pain*, in spite of the most violent spasms, seems not to occur, or but rarely.

Sensitiveness of single vertebræ to pressure, found and emphasized by Stiebel, is not only without doubt very rare (for experienced authors, as Hasse and others, were unable to find it), but could hardly claim any real importance even if it were more common; the many examinations into that which is called spinal irritation have shown how frequent and how destitute of significance this phenomenon is, especially in nervous and hysterical persons.

As to *vaso-motor and secretory disturbances*, I have not been able to discover them. Nothing whatever is said by authors about them.

Psychical disturbances are rarely absent, although in the lighter cases they are but little developed, and consist only of irritability and changeability of temper. In the severer cases, especially when of long duration, the character seems to change for the worse: good-natured persons become passionate; the peaceable, quarrelsome; the cheerful, timid and recluse; the pious lose their scruples and are disobedient; the intelligent appear childish and simple. In connection with the stupid look and the apish grimacing behavior, these signs of depressed intelligence and altered character, especially when occurring in older children at and after the period of puberty, are apt to terrify the non-medical mind; and the physician is often obliged to reassure the relatives, who apprehend that idiocy will be the result. Hasse is inclined to derive the frequent weakness of mind, the want of attention, the failure of memory, the incapacity for orderly thought and logical judgment, from the anæmia, the general bodily weakness, and the continual diversion of thought caused by the muscular contractions. I should prefer, however, the view which derives them from slight anatomical changes in the central apparatus of the nervous system, especially since intelligent patients of the upper classes, who have passed the period of childhood, require a considerable time after the cure of the chorea before they wholly regain their mental powers; and since significant changes have recently been found in the brain in certain cases of chorea (see further on).

The explanation of these facts must rest upon future investigations.

It is but rarely that the psychical disturbances persist or lead to general paralysis (cf. Results).

Apparatus of respiration and circulation.—Besides the choreic disturbances of the respiratory muscles above mentioned, no morbid symptoms, upon the part of the respiratory organs, have been observed.

As regards the organs of circulation, the doubtful arhythmia of the action of the heart has already been spoken of. An acceleration of the pulse at the height of the disorder is quite constant, and may probably be explained as due to the continual muscular effort and the mental disturbance. With the improvement in the symptoms of spasm the frequency of the pulse diminishes.

In a girl of ten, observed by me, who entered the medical clinic at Erlangen at the end of the third week of an attack of hemichorea, and was discharged nearly cured after seventeen days' treatment with arsenic and lukewarm baths, followed by shower-baths, the rate of the pulse was taken twice a day in bed. It averaged 89 for the first six days, in the second period of six days 83, in the last five days 76. The maximum and minimum for the first period were 104 and 80, for the second 88 and 76, for the third 80 and 72.

Physical examination of the *heart* often reveals anomalies, which depend partly upon the residual consequences of an old endocarditis, and are partly of a purely functional nature. The former are characterized by valvular murmurs and by the phenomena consecutive to the valvular disturbances in question; the latter only by a systolic valvular murmur at the mitral, sometimes in the form of a soft-blowing sound, sometimes a harsh sound, or even a distinctly marked rough murmur, although there be no increase in the second pulmonary sound, no enlargement of the right ventricle, nor any other sign of increased tension demonstrable in the system of the pulmonary artery. In a few cases, when the second pulmonary sound is moderately accented, it may be very hard to decide whether there exists an anatomical or only a functional disturbance of the valve. The pathogenesis of these functional disturbances of the mitral in chorea is quite as obscure as in the cases of anæmia, chlorosis, acute rheumatism, etc. That the papillary muscles are the primary cause of the murmur, or the change in the valvular sound, is, *à priori*, more likely in chorea than in any other affection. As to the frequency of the coexistence of endocarditis and its consequences with chorea, see below, under the heading "Anatomical, Experimental, and Theoretical Considerations."

The *bodily heat* is not changed, even in severe cases, in spite of the restless muscular activity. I have carefully tested this question in a few cases, and when there has been no complication, have never found a temperature above 38° C. (100.5° F.) in the rectum. In chorea, only rectal temperatures can be made practically useful, for the motion of the body makes the determination of the axillary untrustworthy, or even quite impossible.

In the above case of hemichorea, treated for seventeen days in the clinic, the rectal temperature was noted twice a day. The maximum reached was in three evening measurements, equalling 38.0°; the minimum in two cases of morning temperature, 37.0°. All the other temperatures ranged from 37.2° to 37.9° C. (99° to 100.3° F.).

The *nutrition* of the patient is as good as could be wished in slight and brief cases, especially when they are taken from school and work, and brought nearer to Nature, in the country or at the sea-shore. In severe cases the appetite and digestion suffer in proportion to the duration of the disease, the patient emaciates, loses in weight,[1] grows anæmic and feeble, and gets a dry cracking skin. The perpetual friction very often gives rise to inflammatory conditions of the skin (excoriations, eczemas, furuncles).

The *stools* are usually rather constipated than the reverse.

As regards the state of the *urine* there is much variety of statement. Its quality is generally noted as normal, its color is either light or dark. In respect to its chemical composition, Bence Jones states that an increased excretion of urea is constant. Stiebel and Veghelm speak of absence of lime, and others of abundance of urates as frequent. C. H. Jones found that the excretion of urea and phosphoric acid was often increased at the height of the disease, and diminished during convalescence. L. Tait observed in a fatal case, in a pregnant woman, much sugar in the urine, with a specific gravity of 1031, no albumen, and a diminution of the chlorides.

Course, Duration, and Results.

Chorea is always *chronic.* The lightest cases, lasting three or four weeks, have been spoken of as acute, and Thilenius has accordingly distinguished an *acute* and a *chronic* form. But the circumstances alleged in support of this view, namely, that the duration is limited to three or four weeks, that all the muscles are attacked at once, and that the disease is fully developed from its beginning, cannot be considered, as Wicke remarks (l. c., p. 274), a sufficient ground for making the distinction.

Nor does it seem necessary to make the division into *stages*, which Hamilton and Dalgairns have attempted. The division into increase, acme, and decrease does not exist in nature, and must be left entirely to the judgment of the observer, and in the numerous cases where there are no prodroma at all, it is, of course, impossible to make a stadium prodromorum.

The *duration* is extremely variable. We find all sorts of figures given, from a few weeks up to many years. Wicke obtained an average duration of 89 days from 125 cases, of which the duration was accurately known; Sée, an average of 69 days from 117 cases.

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[1] In a case observed by me, a girl of eleven had fallen in weight at the severest period of her sickness to 55.5 lbs., and during convalescence rose to 61 lbs. in ten days, making a gain of 5.5 lbs.

According to Moynier's statement (l. c.) the duration was different in the two sexes, being in girls from 33 to 37 days, in boys from 74 to 81 days. But it can hardly be believed, in opposition to all the other authorities, that so considerable a difference should exist. My own views are decidedly opposed to those of Moynier, for I have repeatedly observed the disease to last four or five months in girls.

Gray and Tuckwell observed the duration of six and twelve cases respectively, under purely expectant treatment. Gray found the longest duration twenty weeks, the shortest six; Tuckwell, the longest seventeen, the shortest six. The average of all was ten weeks, and one and three days respectively. Hillier also obtains an average duration of ten weeks from thirty cases. Here is the place to observe that Tuckwell found almost the same average duration, namely, ten weeks and six days (from seven to sixteen weeks) in eight children, who were treated after Begbie's method, by increasing doses of arsenic, and that Gray found an average duration of ten weeks and five days in seven cases treated with various remedies.

In general, therefore, the duration of chorea may be considered as from two to three months; but this statement must not be taken as giving a trustworthy indication of the probable duration of an individual case.

Cases in which chorea lasts many years, or a whole lifetime, are usually symptomatic, and are chiefly based upon anatomical lesions of the brain and cord (Cyon's chorea symptomatica; Sée's chorée chronique in the stricter sense, as distinguished from his chorée vulgaire).

The assumption that chorea has a *cyclic course* (is self-limited),[1] as stated by Gintrac, with a duration of at least two and at most three months, is somewhat arbitrary, and stands opposed to the experience of every busy physician, but has had the important practical result that all treatment, except the dietetic, has been declared superfluous and useless. But in most severe cases in which the symptoms last longer than three months with unabated severity, it is easy to convince oneself of the incorrectness of the cyclic theory, and of the possibility of quickly influencing the disease by therapeutic measures. I have had the opportunity almost every semester, in my clinic at Erlangen, of presenting instructive cases of this nature (compare Treatment of Chorea).

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[1] Journal de la société de méd. de Bordeaux. Avril, 1845.

The course is seldom quite uniform, even when the disease has reached its height. Improvement alternates with relapse, and psychical affections of comparatively small importance affect the violence of the symptoms very much. Such relapses in consequence of psychical excitement are also very common during the period of convalescence.

Recurrence of the disease is frequent, and occurs at the greatest variety of intervals. This may be due to violent psychical excitement, or bodily conditions, such as disease or pregnancy, acting on a person who is clearly predisposed. Most of the relapses occur during puberty, but among persons who were choreic at or before this period they are observed at from twenty to thirty years of life and later.

The number of recurrences varies. Most frequently it happens but once; twice is rarer, and more than twice is still less frequent. Sée observed thirty-seven among one hundred and fifty-eight cases; among these, seventeen had but one relapse, thirteen had two, and six had three; in one case seven sharply marked attacks could be shown. Cases of six relapses are given by Rufz and Romberg. When they are so numerous the intervals between them are usually but short.

Whether the *season of the year* can influence the occurrence of relapses, as Wicke believes, is a matter which requires further proof. Among thirty-five relapses, thirteen are stated by Wicke to have occurred in spring, twelve in winter, nine in autumn, and one in summer.

As to the *termination* of the disease, a complete cure is by far the most frequent. Trustworthy statistics, such as are furnished in many other points by hospital observation, are not to be had, for the patient's condition after discharge (which usually takes place at an early period), and such relapses as may occur, usually remain unknown. Slight disturbances of coördination of movement, especially in the upper extremities and the face, needless haste in executing certain motions, grimaces, etc., often remain for years after an apparently perfect cure, and in a few cases never disappear.

Termination by *incomplete cure* is rare. Either a few symptoms of chorea itself may persist, or pareses or paralyses may develop in the half of the body which has been more affected, or else a permanent psychosis is developed in the course of the disease: as mania, melancholia, progressive paralysis; but these events are very rare in uncomplicated chorea. Usually there is some cerebral lesion before the chorea commences, and the latter is only a transitory phenomenon which gives way to other and severer disturbances.

Death is extremely rare in children, especially in uncomplicated cases. In adults, and especially in pregnant women, it is much more common. Sée found nine deaths among one hundred and fifty-eight cases in the children's hospital, or 5.7 per cent. The statistics of Wenzel give a mortality in chorea gravidarum of 27.3 per cent. Of sixty-four pregnant women, and two women lying-in who were attacked just after delivery, eighteen died. Of these eighteen, the number of the pregnancy is noted in eleven, of whom six were primiparæ, four in their second confinement, and one in her fourth.

The cause of death is to be assigned, on the one hand, to the intensity of the disease itself; on the other, to many serious affections and complications, as meningitis, encephalitis, myelitis, pericarditis, endocarditis, and embolic processes.

In cases which seem to be caused by the chorea itself, the symptoms are usually violent from the first and in a few days rise to an enormous severity, which is succeeded by sudden collapse, and death follows after more or less of coma, involuntary stools, etc. The choreic twitchings either cease entirely when the collapse begins, or, in a few cases, they continue, but grow weaker, until death.

Anatomical, Experimental, and Theoretical Considerations.

The scanty pathologico-anatomical data which we possess are chiefly drawn from late research. The older autopsies, collected by Wicke and Sée, are nearly useless in determining the nature of chorea, both because the error commonly made in confounding chorea with the so-called chorea major and paralysis agitans justifies a doubt as to the correctness of the diagnosis, and because the inferior knowledge and methods of pathological anatomy previous to the year 1840 confounded essentials with non-essentials in the points observed. In particular, a careful distinction between old lesions and those which might be associated with chorea, and a minute and microscopical examination, are wanting. Hasse has stated that along with the positive results (doubtful as to their interpretation), which consist of hyperæmia and inflammatory exudations of the membranes of the brain—more rarely those of the cord,—local softening and other local diseases of the brain or cord, new formations, etc., there existed a number of entirely negative reports.

Sée, who compares the autopsies made before his time with his own, finds among 84, 16 with results entirely negative in respect to the nervous system; 34 with inflammation of serous membranes, changes in the heart, etc. (16 arthritis rheum. multipl., 11 periendocarditis, 12 pericarditis and endocarditis, 6 hypertrophy of the heart, 6 meningitis, 3 bony indurations of the meninges, 6 pleural exudations, 6 peritonitis); and 32 in which there were affections of the central nervous system and its membranes, oftenest softening and tuberculosis.

Although Sée recognized correctly the close relationship which chorea bears to rheumatism and endocarditis, he yet thought that only a minority of the deaths should be referred to inflammatory changes in the heart, and the majority to the action of chloro-anæmia and nervous excitement.

Roger, on the contrary, maintains that the connection between chorea and disease of the heart is neither rare nor accidental, but is just as regular as the connection between rheumatism and affections of the heart. The acute rheumatism of the joints, the affection of the heart, and the chorea he makes but three manifestations of the same diathesis. The order in which they appear is very variable. The rheumatic diathesis can also develop from scarlatina. Among 71 cases of his "chorea cardiaca," Roger found simple endocarditis forty-seven times, endopericarditis nineteen times, simple pericarditis five times. But it should be added that these figures are chiefly based on diagnoses made during life, and that a systolic apex murmur seems in France to justify the inference that endocarditis is present.

Roger's too general statement was soon objected to by the more judicious of his own countrymen; Trousseau declared that in his own experience rheumatism and chorea often accompanied or succeeded one another, but not always.

In Germany and Austria the connection between rheumatism and chorea would seem to be much less close, and certainly not to constitute a law in Roger's sense. Steiner, at least, found only four cases out of 252 observed by him in Prague, in which the chorea had developed from acute articular rheumatism. These numbers seem extremely low; for my own part, I have found about one case in five in which it could be proved that rheumatism had preceded the attack.

According to the observations of English authors, less importance should be attached to the rheumatism than to the endocarditis and its sequelæ, which are very often found in the autopsy of choreic children, without previous acute rheumatism. The English reports show, beyond a doubt, that in that country delicate vegetations on the mitral valve, less frequently on the aortic valve also, are very commonly found in the bodies of choreic patients.

We will extract only a few data.

Ogle found in sixteen autopsies of chorea "fibrinous, membranous, or granular deposits upon the endocardium of the valves" ten times; also pericarditic changes three times, congestion of the central nervous organs six times, and softening of the cord once.

Pye-Smith describes four deaths among 150 cases of chorea, observed in Guy's Hospital and the Metropolitan Free Hospital during the past three years, and adds seven other reports of autopsies not yet published. Of the 11 cases, 2 were in males and 9 in females; the ages of the former were 10 and 38, of the latter, 7, 7½, 12, 13, 16, 18 (twice); the age of one pregnant woman is not given. In all eleven cases recent or old endocarditic products were found upon the valves (valvular vegetation, with and without coagula, on the mitral alone five times, on the aortic valves alone twice, on both at once four times). Twice in the eleven cases the heart was hypertrophied (once on the left, once on both sides). Cerebral hyperæmia is noted three times, hyperæmia of the spinal cord once, hydrocephalus, with a corresponding flattening of the gyri three times, pericarditis once, pleuro-pneumonia three times, diphtheria twice. (It is matter for regret that no statements are made respecting the results of microscopic examination.)

In England the doctrine that chorea is a cerebral disturbance has found warm supporters since the beginning of the present century. Of later authors Todd was the first to point to the importance of unilateral chorea and its not infrequent combination with hemiplegia as indicating a cerebral origin; he has pointed out the results of comparative weighings of parts of the brain, made by Aitken in the case of a patient dead of chorea. These gave the specific weight of the corpus striatum and thalamus of the right side as 1.025, on the left 1.031, while Bucknill's weighings of healthy brains give a weight of 1.036 for these organs. By the side of these doubtful proofs, all the more value is to be attached to the careful anatomical examinations to which some English observers have devoted themselves with special zeal and success. In particular, the names of Kirkes, Broadbent, Tuckwell, Ogle, Hughlings Jackson, Barnes, Russel ought to be mentioned.

Kirkes endeavored in 1850 to prove that inflammatory changes in the valves of the heart were the usual change found in autopsies of fatal chorea, and took a considerable part in the causation of choreic symptoms. Rheumatic affections were in very many cases not demonstrable, and yet endocarditic processes were shown by examinations made during life and after death; the latter were sometimes found after death, when no cardiac murmurs had been audible during life. The opinion of Begbie, Watson, Branson, and others, that a "rheumatic diathesis" is the common cause of affections of the joints, endocarditis and chorea, Kirkes could not regard as satisfactory, as chorea and valvular diseases often coincided without the least trace of a rheumatic tendency. He considers it most probable that *endo-*

carditis is the cause of the chorea, through the inflammatory products of the valves which become mixed with the blood and disturb the functions of the nervous centres.

Broadbent[1] has used numerous autopsies as means for carefully formulating the cerebral nature of choreic disturbances, and first designated the corpus striatum and thalamus opticus as their location. A variety of morbid states of these ganglia might produce the group of symptoms known as chorea; but the most frequent, in his experience, was *capillary embolism of the corpus striatum, thalamus opticus, and their vicinity.*

According to the extent of the embolism, chorea is of greater or less extent or intensity, and may be combined with delirium, mania, etc. Besides embolism, other sorts of disturbances of nutrition in the ganglia, peripheral influences which arrest the reflex process, direct lesion of the ganglia from fright, mechanical lesion, etc., may act as causes.

These views have been adopted in the main by the other English neuro-pathologists, and continual attempts have been made to confirm them by anatomical proofs.

Tuckwell reports the autopsy of a girl aged thirteen, in whom chorea developed out of rheumatic fever, and in three months led to death by exhaustion, without mania or delirium.

At the autopsy there was found *softening of the right middle cerebral lobe, and to a less extent of the left, without demonstrable embolism.* The grey cortex was intact upon the surface, but the deeper layers, and in particular the adjacent white substance as far as the (intact) corpora striata and thalami, were changed. Under the microscope, the walls of the small vessels were covered with fine granular punctations; the granules were in some places scattered, in others heaped together.

On the *auricular surface of the mitral there were numerous fine warty vegetations.* In the kidneys three small arterial twigs were plugged with emboli.

In a case observed by Tuckwell before death, this author was enabled to demonstrate embolic processes as a cause of the softening of the cerebral convolutions. The vegetations, which, as a rule, are situated on the auricular surface of the mitral, are remarkable for their smallness and delicacy, and can be easily removed with a brush, and doubtless also by the current of the blood.

Very similar is the condition found in a case of acute chorea, observed by Gray:

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[1] The article cited from the British Med. Journal, in the list of authorities, is a development of a lecture delivered by *Broadbent* before the Medical Society in London, in the year 1865-6, but not published.

embolic thrombosis of the basilar artery, both vertebrals, and both middle cerebrals, with softening of the anterior and middle cerebral lobes and the dorsal part of the cord.

Microscopic embolism of the corpus striatum and small vegetations on the mitral valves were demonstrated by E. L. Fox in a case of acute chorea, in which the patient died twelve hours after the occurrence of a cerebral hemorrhage.

Granule-cells along the vessels in the corpus striatum have been observed by numerous authors (Tuckwell, Ogle, etc.).

Germany has made but few contributions to the pathological anatomy of chorea within the last twenty years.

Froriep examined the body of a boy of ten who had suffered over a year from chorea, and found an impression upon the lower (anterior) surface of the medulla oblongata, caused by a considerable enlargement of the odontoid process.[1]

Romberg[2] describes three autopsies; one showing a general atrophy of the brain with hydrocephalus externus; one internal hydrocephalus with softening in the neighborhood of the ventricles; and a third with softening of the cervical and dorsal part of the spinal cord.

Of two interesting cases of reflex chorea from Frerichs' clinic,[3] one was examined post mortem.

The case was that of a pregnant woman, and the chorea was complicated with mania. The birth of a dead child occurred about the thirty-third week of pregnancy. In spite of this, the choreic movements continued with increasing violence until death, which took place a week after delivery. The autopsy showed a general hyperæmia of the brain and its membranes, pachymeningitis interna vascul. et hæmorrhagica, deposition of bone upon the dura mater, warty excrescences on the auricular aspect of the mitral, and a remarkably narrow aorta.

Very valuable anatomical researches, especially of a pathological and histological nature, have been made by some Austrian observers.

Rokitansky[4] first pointed out the occurrence of interstitial growths of connective tissue in the central nervous system in chorea.

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[1] Neue Notizen a. d. Geb. der Natur- und Heilkunde. No. 224. p. 57.

[2] Nervenkrankheiten. p. 528 seq.

[3] Dissertations of *Arnoldi* and *Fricke*.

[4] Ueber die Bindegewebswucherung, etc. Sitzungsberichte der Wiener Akademie der Wiss. 1857.

Steiner[1] was enabled to confirm the existence of hyperplasia of connective tissue in the cord in one case; with it there was a serous exudation and effusion of blood in the spinal canal; in a second case he found hyperæmia of the brain and spinal cord, serous effusions within the spinal canal, and hemorrhage at the upper part at the exit of the nerves; in a third case the remains of acute rheumatism of the joints and endopericarditis with a turbid serous exudation in the spinal canal.

Meynert[2] made a microscopical examination of the brain and cord in one case and found the principal anatomical changes in the gray substance of the central ganglia, extending thence to the cortex of the brain. The changes consisted in processes of division in the nuclei of the nerve-cells, interstitial growth of nuclei, and hyaline swelling of the nerve-cells with molecular decay of the protoplasm. In the cord he found swelling of the *Saftzellen* of the reticulum [star-shaped interstitial connective-tissue corpuscles].

Very remarkable results were found at Elischer's[3] examination of the nervous system of a parturient woman with chorea, who died at Prof. Korányi's clinic at Budapest. The account of the disease presents an excellent instance of chorea gravidarum et puerperarum.

A. Cz., æt. twenty-two, laboring woman, was said to have had chorea in her eighth year; and in her sixteenth she had two attacks, the first light, the second severe, confining her for six weeks in the children's hospital.

First pregnancy, 1870: chorea reappeared in the sixth month, lasting till delivery (which occurred normally), and then ceasing.

Second pregnancy, 1872: reappearance of chorea in the fifth month (December). No relief from treatment. Admitted to Prof. Korányi's clinic, February 23, 1873. Patient emaciated and pale. Bronchitis of the right side. Heart normal. Excessive unrest of the muscles, worse on the right than on the left side. Constipation and retention of urine. Can only eat and drink by artificial means, and with the greatest precautions.

February 28, a child still-born in the night. The spasms are somewhat relieved. On the 29th, violent spasms. Some rest is obtained by small doses of chloral hydrate (0.66 gram.) On the succeeding days endometritis sets in, the spasms increase enormously, and death occurs on the 3d of March.

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[1] Prag. Vierteljahrschr. 1868. Jahrg. 25. Bd. III. p. 45 seq.

[2] Allgem. Wiener med. Zeitung. Nos. 8 and 9. 1868.

[3] Virchow's Archiv. Bd. LXI. p. 485. 1874, and Bd. LXIII. p. 104. 1875.

Autopsy: Hyperæmia and œdema of the brain and the gray substance of the spinal cord; hypostatic pneumonia of the left, bronchitis of the right lung; partial steatosis of the liver; hemorrhagic erosions of the stomach, cloudy swelling of the epithelium of the kidney, puerperal peritonitis.

The microscopical examination embraced the brain, the cord, and, of the peripheral nerves, the right median and sciatic. These nerves are found lessened in size,[1] flattened, of abnormal hardness, and of a dirty gray color. Under the microscope the connective tissue appears excessively developed, very rich in granulated nuclei, and in places traversed by small extravasations from the very full vasa nutrientia. The medullary sheaths are, in some places, as it were, covered with hoar frost; in others in a state of glassy swelling, apparently of softer consistency, their axis cylinders not visible, or only slightly shown by a darker tinge of color.

Spinal cord.—Membranes hard and much congested. Abundant proliferation of nuclei in the adventitia of the vessels. In the central canal is contained serum, and the web of connective tissue surrounding it is harder than usual. In the gray substance the four large groups of ganglion cells are distinctly marked off by encircling lines of connective tissue. Abundant development of nuclei in this connective tissue, most abundant in the substantia gelatinosa Rolandi. The ganglion cells have a coarse, lumpy look, are indifferent to carmine (only their processes taking a slight tinge), are rusty yellow, contain lumps of pigment, and are without visible nuclei. The white substance of the anterior columns is relatively intact, the axis cylinders still easily distinguished.

The *brain* also showed *regressive metamorphosis* (fatty, amyloid, and pigmentary change) of the nerve-elements and vessels of the large central ganglia, the island of Reil and the claustrum, with very small secondary extravasations of blood in the connective tissue, numerous emboli in the smallest vessels, especially in the cortex. The division of the nuclei of the ganglion cells, found by Meynert, could be made out by E. only in the formation of the claustrum. The granule cells along the vessels of the corpus striatum, upon which Tuckwell laid so much stress, did not seem important to E., as he had found them in other brains, *e. g.*, in that of a tuberculous person, and that of a person who had bled to death. He leaves it undecided whether some of the changes found by him should not be referred to the puerperal disease.

According to Elischer, the appearances of the peripheral nerves, the spinal cord, and the brain indicate that the morbid process is not confined to one organ, but is *diffuse*, and depends on *irritative processes*. In favor of the latter view, he mentions the proliferation of nuclei in the connective tissue of the peripheral nerves and along the vessels of the cord, the thickening and the deposits of lime in the adventitia of the vessels in the thalamus opticus and corpus striatum, the processes of deposition upon certain portions of the intima, and, equally important, the proliferation of cells in the formation of the claustrum, and the single pericellular nuclei.

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[1] At the point where the sciatic left the ischiatic foramen, E. counted only thirteen bundles of nerves instead of twenty, which is the number known to be usual.

The irritative processes are followed, furthermore, by those regressive metamorphoses which are most distinctly marked in the vascular system.

According to Elischer, we are entitled to conclude that all the changes proceed from *one source of irritation*, which acts diffusely upon all the nerve trunks, and reaches a high degree of activity in only a few spots, where it produces more decided alterations.

Charcot has expressed himself very recently as in favor of the cerebral nature of chorea, basing his opinion upon three autopsies from cases of hemichorea after hemiplegia (chorée post-hémiplégique), of which he observed five or six. There existed anæsthesia of the skin and organs of sense during life, upon the paralyzed side. At the autopsies ochre-colored cicatrices were found at the posterior end of the thalamus opticus and the nucleus caudatus, and at the hindmost portion of the foot of the corona radiata. In two cases the anterior corpus quadrigeminum of the affected side was involved. Charcot expresses the suspicion that the changes which caused the chorea were located in motor fibres, situated in front and to the side of the fibres of the corona radiata, which preside over the conduction of sensitive impressions.

Charcot has also observed cases in which hemichorea with anæsthesia came first, and hemiplegia followed; and in one such case he found a collection of blood, as large as a nut, in the posterior half of the thalamus opticus. Tumors of this region may cause hemichorea, according to Charcot, which may be followed by hemiplegia or may not. Finally, Charcot has seen hemichorea caused in children by partial atrophy of the brain.

The following report of an autopsy, given by Camillo Golgi,[1] is important, although complicated with mental disorder.

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[1] Rivista clinica. 1874. IV. p. 361

The case is that of a teacher, whose mother was hysterical, and who had been devoted to Bacchus and Venus from early life. In his thirty-second year he was attacked by chorea, with maniacal excitement. He passed the following years partly in insane asylums, partly with his family. No change occurred in the chorea, but the power of his mind diminished perceptibly. Ten years after the commencement of his disease he entered the hospital for incurables at Abbiategrosso. The attendant physician found no group of the voluntary muscles free from chorea. He was forgetful, suspicious, indifferent to relatives and friends, destitute of sexual appetite. There were alternations of fury and quiet, of voraciousness and abstinence. Death from pneumonia followed soon after his entrance.

Autopsy.—Pachymeningitic membrane on the right cerebral hempishere; dura mater everywhere thickened, pia mater the same; meningeal vessels crowded with blood. Convolutions of the great hemisphere in part atrophic; the medullary substance infiltrated with serum, softened, hyperæmic, lateral ventricles full of cloudy serum, ependyma thickened. Corpora striata much changed, consistency less, glistening red color, and a general gelatinous look. Spots of gray degeneration at the outer side of the corpora striata and in the centrum semiovale.

In the cerebellum nothing but congestion of the vessels and diminution of consistency.

Spinal cord.—Membranes thickened, medulla strikingly soft, boundary between the two substances of cord not sharp.

The microscopical examination showed, in the affected convolutions (frontal and parietal), thickening of the vascular walls, with abundance of nuclei, and a great many lymph corpuscles in the perivascular lymph-spaces. The ganglion-cells presented the greatest variety of changes, being small, humpy (höckrig), hard to isolate, and without reaction to one per cent. solution of silver. Their protoplasma processes were humpy, convoluted, thinned. The axis cylinders were lessened in diameter, thickened in knots, and convoluted. The same change in the ganglion-cells was found in the superficial layers of the corpora striata. There was also an extensive cretification, with fatty degeneration and pigmentation of the ganglion-cells. The same cretification in Purkinje's cells of the cerebellum.

In the spinal cord, increase of the connective tissue, amyloid corpuscles in the medullary substance, sclerosis of the posterior and lateral columns in the cervical region. In the gray substance the cell-bodies were strongly pigmented and easily broken down.

Golgi regards these alterations as the results of a *chronic interstitial encephalitis*, and lays emphasis upon the great similarity between these histological changes and those found in dementia paralytica.

The close connection between chorea and the true psychoses and epilepsy has been pointed out by Tommasi, Wilks, Arndt, C. H. Jones, Clouston, L. Meyer, Leidesdorf, Russel, and others, and numerous observations of the coincidence of chorea with mental disorder (less frequently epilepsy) in the same individual have been produced.

Tommasi describes a case in which the symptoms of chorea alternated with those of dementia paralytica.

According to Russel, mental disturbance accompanies, not only severe, but often slight attacks of chorea. Among thirty-eight cases with changes in the intellect and feelings, he observed six severe cases with delirium and mania. In three of these cases the psychical and the choreic changes were manifestly independent of each other, and should be taken as co-effects of the same cause; for in one case the psychical alienation appeared *before* the choreic symptoms, and in another it continued several weeks after the muscular twitchings had ceased.

According to Leidesdorf, these frequent changes in disposition and character are not seldom accompanied by feelings of anxiety and hallucinations of the senses, particularly that of sight, which latter commonly appear at the moment of falling asleep, and greatly distress the patients by their frightful nature. Leidesdorf saw maniacal attacks chiefly in those cases of chorea in which the latter had developed from a febrile complaint.

Hallucinations of feeling, hearing, smell, and apprehension of being poisoned, with great confusion of mind, were observed by Ritti[1] in a case of chorea, in which, after long-continued prodromic symptoms, the choreic twitchings began in the fingers and extended to other parts. Recovery in two months.

In the *spinal cord* alterations have been repeatedly found, namely, hyperæmia of the medulla and the membranes, softening of the cervical and also of the dorsal medulla (Romberg, Ogle, Gray, Golgi, De Beauvais, Hine, Brown-Séquard, Lockhart Clarke), interstitial proliferation of nuclei and hyperplasia (Rokitansky, Steiner, Meynert, Elischer), and sometimes serous exudation in the central canal, proliferation of nuclei in the adventitia of the vessels, and regressive metamorphosis in the ganglion-cells (Elischer).

The *peripheral nerves* have received attention only in Elischer's researches. He found, as above stated, the vasa nutrientia crowded with blood, and small extravasations in places; the connective tissue between the nerve bundles greatly developed, very rich in nuclei; the medullary sheaths of the nerve-fibres in places covered as it were with frost, in other places in a state of vitreous tumefaction, seemingly softer than normal, while their axis cylinders had become invisible in great part. Both the nerves examined (the median and the sciatic) seemed to the naked eye small, flat (abnormally), and of a dirty gray color.

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[1] Union méd CXXXII. 1873. Virchow-Hirsch, Jahresber. 1873. II. 1. S. 42.

Before passing on to formulate a summary of the nature of chorea, based on the previous statements, we have to speak of a small number of important *experimental investigations* made by French authors. The object of these has been to settle the question as to the portion of the nervous system attacked by the cause of irritation, and what special part was taken by the spinal cord in chorea.

Chauveau[1] took dogs suffering from general chorea, cut the spinal cord high up close to the skull, and found that the choreic movements lasted for hours, until the death of the animals. Section of the dorsal medulla puts an immediate stop to the twitchings in the muscles of the tail and hind legs. Chauveau concludes from these experiments that neither the cerebrum nor the cerebellum is the point of origin of chorea, but that the spinal cord is the seat of irritation.

Carville and Bert, with the same experiments, reached the same results.

Legros and Onimus[2] studied the muscular twitchings of choreic dogs by the graphic method. They found that each individual contraction occurs brusquely, but passes off more slowly, often intermittingly, and is followed by a period of repose. They also observed repose following the injection of chloral hydrate (3.50 gram.) into the rectum. Section of the cord at the skull left the contractions undiminished as long as life was kept up (three or four hours) by artificial respiration.

Irritation of the posterior columns of the exposed spinal cord with a scalpel increased the twitchings. Section of the posterior roots had no influence, as Bert found. Partial removal of the posterior cornua and columns was followed by a weakening, but deeper incisions by a cessation of the movements. Legros and Onimus conclude from this, that *the nerve-cells of the posterior cornua, or the nerve-fibres which unite them with the motor cells, are the seat of chorea.*

These observers found a striking difference in the action of the constant current, dependent on the direction in which it was passed. An ascending current increased the intensity and the frequency of the contractions, while a descending current weakened them considerably. If they had been reduced by poison or by the weakness of the animal, an ascending current was able to restore them in their old intensity. These effects occurred even when the electrodes did not directly touch the spinal marrow, but were laid upon the uninjured skin, provided the direction was retained. Mechanical or faradic irritation of the posterior roots was followed by a considerable increase of the movements after the stimulus was withdrawn.

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[1] Archives gén. de méd. Mars, 1866.

[2] Recherches sur les mouvements choréiformes du chien. Comptes rendus. Tom. LX. 1870. p. 1046.

One more experiment remains to be mentioned, contributed by M. Rosenthal (l. c., p. 579) from the Vienna Institut für experimentelle Pathologie. A dog with choreic movements of the right fore-leg was taken, and fine flower-seeds injected into his left carotid. Although the voluntary movements instantly and completely ceased, the twitchings became strikingly stronger in the anterior extremities, the eyelids, and the tail, and lasted two days, until the animal's death. The autopsy showed encephalitis of the left anterior lobe, softening of the left corpus striatum, embolism of the left arteria fossæ Sylvii. Microscopical examination showed spots of proliferation of connective tissue in several parts of the brain substance. Rosenthal infers from this experiment that the artificial disturbance of the circulation in the brain, with loss of the influence of the motor ganglia, produced an increase in the choreic movements, probably through irritation of the centres of co-ordination in the middle brain and cerebellum. It seems to me that this experiment, as related by Rosenthal, permits of no definite conclusions.

The scientific material contained in the above presents us with no constant anatomical changes in chorea, and fails to give a perfectly clear view of the nature of the process, but it offers very important points in localizing the process in the nervous system, and in explaining its pathogenesis. In especial, it has been shown by the studies of histological changes quoted from Meynert, Elischer, and others, that we may hope in this way to reach a better understanding of the matter. Doubtless a much larger number of observations and careful autopsies is needed before we can safely allow ourselves to make further conclusions. Let us here recapitulate the most important points.

As regards the *seat* of the disturbances, the anatomical and histological investigations of recent date point first of all to the *brain* as the chief, though not the exclusive, seat of the anatomical change, but show that the spinal cord and the peripheral nerves may also be drawn within the circle of changes. The process, as it seems, is chiefly confined to the gray substance both of the great basal ganglia and of the cortex, and bears the anatomical character of irritation and its consequences, regressive metamorphosis of nerve-elements and interstitial hyperplasia. These irritative processes with their results may, as it seems, remain confined to single regions of the cerebrum, *e. g.*, to one hemisphere or even portions of the hemisphere, especially the corpus striatum, the thalamus opticus, the posterior striæ of the corona radiata, or may be extended diffusely over the whole brain, preferring the gray substance, at the same time attacking the spinal cord and the peripheral nervous system.

That the cerebrum and, above all, the great basal ganglia form the chief seat of the changes in chorea, can hardly be doubted in the present state of the facts. Not only the statements of anatomical histology, but the clinical facts as well support this view. Of especial importance are the frequency with which the choreic symptoms are unilateral, sometimes in association with anæsthesia of the skin, and the transition (although uncommon) from hemichorea to hemiplegia, and the converse process of the development of hemichorea from unilateral palsy (Charcot, Foote, Weir Mitchell, Hughlings Jackson); also the occurrence of unilateral facial palsy, bearing the marks of cerebral origin, appearing at the same time and on the same side with the chorea, and disappearing with it (Broadbent), the frequent coincidence of the chorea with disturbances of mind or disposition in all degrees, from the slightest mental indisposition to the severest forms of mania and dementia paralytica, and the development of chorea subsequent to inflammatory processes in the contents of the skull (encephalitis, meningitis, tumors).

In presence of these facts we cannot attribute a decisive value to the experiments of Chauveau, Carville, Bert, Legros and Onimus, which are believed to show that the choreic changes are situated not in the brain but in the spinal cord. We believe that these experiments only render it probable that in choreic dogs the spinal cord, *i. e.*, the gray substance of the posterior cornua, plays an essential part in producing the phenomena, but do not prove that the brain is quite unaffected, and that the case is the same in the human species. And still, the numerous autopsies in which softening of the cervical cord, or hyperæmia of the cord and its envelopes is found, and the rare cases of embolic processes in the cervical cord (Tuckwell), or interstitial proliferation of nuclei and connective tissue (Rokitansky, Steiner, Meynert, Elischer), render it very probable that the spinal cord plays a part in chorea in man also. How frequent these changes may be in proportion to those of the brain is a question which cannot be answered until we have a fuller series of careful examinations of the pathological histology of the central nervous organs. The same is true of the changes found by Elischer in peripheral nerves. It is reserved for those observers who shall have the opportunity of examining the bodies of patients who die in chorea, to follow out the changes in the nerves given by Elischer.

A question quite as hard to answer as the preceding is that as to the *kinds of irritation* which are capable of producing, or which are required to produce, such momentous disturbances.

It can hardly be doubted, after the cases which have been published, that embolic processes may act as irritant causes. The enormous frequency of the coincidence of chorea with recent and old vegetations of the valves, which has been placed beyond a doubt by numerous autopsies, permits us to suspect that there exists a causal connection between the endocarditic and the choreic disorder; and anatomical researches have demonstrated to a certainty the occurrence of very small embolic processes in the vessels of the brain, particularly of the corpus striatum and thalamus, and once in those of the cervical part of the spinal cord (Tuckwell). True, it remains doubtful whether the relations of endocarditis and its results to the irritative disturbances of the brain are always such as are due to the production of embolism, or whether there may not be some other sort of connection —perhaps both may be due to one and the same unknown irritation.

The pathogenesis of *reflex chorea* is still more difficult to understand, if we retain the anatomical changes in the brain as the chief basis of chorea. We need only refer to the numerous cases of chorea during pregnancy, which, although a predisposition commonly exists, yet require the processes in the uterus to excite them to action, and quickly cease after delivery; also the cases in which chorea originates in peripheral lesions or cicatricial contractions, and gets speedily well after excision of the latter (Packard).

In regard to these pathogenetic questions we find ourselves in the same position as in regard to those *reflex paralyses* which occur during affections of the intestine, uterus, bladder, or kidney, injuries of peripheral nerves, etc., with this difference, that the obscurity which has hitherto prevailed in regard to the pathogenesis of reflex palsy has been somewhat lessened by the experimental studies of the last ten or twenty years, and especially by the works of Gull,[1] Kussmaul,[2] Leyden,[3] Feinberg,[4] Fischer,[5] and Klemm.[6]

We have at present no actual proof in the case of chorea of an anatomical connection between the peripheral irritation and the lesion of the cord, such as may be considered as proved in many cases of so-called reflex paralysis; but the assumption that an irritative process is propagated from the peripheral nerves to the central organs has obtained some support since Elischer demonstrated definite changes in the peripheral nerves in at least one case of chorea, and since the experiments of Klemm and the clinical observations of Lallemand, Martinet and Leyden established the possibility of a propagation of inflammatory conditions from the peripheral nerves to the brain and the secondary development of encephalitis, etc. It should in these cases be assumed that the irritative processes in chorea, which are propagated from the periphery to the spinal cord and brain, and diffuse themselves in these organs, must be less in degree or different in kind from those of neuritic central paralysis, as their effect is so different.

We have no positive means of explaining the pathogenesis of chorea, when caused by violent psychical impressions (fright, anxiety, etc.); we can only express a suspicion that these cases, as well as the similar ones of epilepsy, depend on nutritive disturbances in the brain, caused indirectly by the violent action of psychical emotions upon the vaso-motor centre, and directly by the anomalous action upon the vascular currents of the brain, which proceeds from that centre.

Guy's Hospital Reports. 1861 and 1862.

Würzb. med. Zeitschrift. Bd. IV. S. 56 ff. 1863.

[3] De paraplegiis urinariis. Königsberg, 1856. Also, Ueber Reflexlähmungen, *Volkmann's* Sammlung klinischer Vorträge. No. 2. 1870.

[4] Ueber Reflexlähmungen. Berlin. klin. Wochenschrift. No. 41. 1871.

[5] Ueber Neuritis. Inaug. Diss. Königsberg, 1869.

[6] Ueber Neuritis migrans. Inaug. Diss. Strasburg, 1874.—Cf. also *Leyden*, Klinik der Nervenkrankheiten. Bd. II. 1. S. 214 ff. Berlin, 1875, and *Erb*, Diseases of the Peripheral Cerebro-spinal Nerves, in this Cyclopædia (volume XI., p. 399 et seq.)

It is manifest that the gravity of the lesions will be very different in the different degrees of intensity of chorea. The autopsies have chiefly been made in severe cases, and in them the degenerative processes were found very considerable. In the light cases, of which the majority recover completely in two or three months without any considerable changes in the psychical and intellectual sphere, only slight and perfectly curable disturbances can be supposed to exist.

The sum of our knowledge of the pathologico-anatomical changes is so small that it is scarcely of consequence to attempt to explain the way in which they act upon the functions of the central nervous system, but I will give in this place the view of one of those best acquainted with chorea, namely Broadbent. He considers that the processes in chorea are always such as merely weaken the force of the nervous apparatus without destroying its structure. Hence, the weakness of the muscular force and the diminution of sensibility, so common in chorea; hence, the frequent termination in paralysis. He gives to the condition of the system the name of "delirium of the sensori-motor ganglia of the brain." In ordinary delirium imperfect ideas are rapidly evolved, and there is no control over the mental processes; in chorea the control over the motor apparatus is wanting. The movements are excessive in number and extent, but without force and precision.

Diagnosis.

With such a remarkable group of symptoms the diagnosis of chorea ought hardly ever to be difficult. In particular, it is hardly possible to confound it with the *tremor* of age, of drinkers, of those poisoned by mercury and lead, or with *paralysis agitans*, or *multiple sclerosis of the brain and cord*, if the physician has had any experience in nervous diseases. The partial spasms of single muscles or groups of muscles in the region of certain nerves, *e. g.*, the accessorius (external branch) and the facialis, and the (Beschäftigungsneurosen) neuroses of co-ordination, connected with the patient's occupation, such as writer's cramp, cannot easily be confounded with chorea if carefully examined; the former occupy definite regions, and remain confined to them, while the latter are produced only by the special action proper to the avocation of the patient, and (at least in the beginning) appear only in the muscles required to perform the action. I therefore think it needless to repeat the characteristic symptoms of chorea in conjunction with those of other neuroses.

Prognosis.

This is decidedly favorable, as a rule. If the disease appears during the period of bodily development, is primary, and without complications—if the contractions are not excessive in severity, if sleep is good, if there are no considerable psychical disturbances and no complications, the prognosis may be set down as favorable. It becomes dubious when the violence of the movements is such as to exhaust the patient, and when they continue during sleep; when very little food can be taken, when there is delirium, collapse, etc. The inactivity of remedies which are usually efficacious may also very properly be taken into account.

In determining the possibility of a relapse, in any given case, we are entirely without resources, except when there is a decided tendency to the disease; hence it is necessary to observe caution in giving an opinion.

The prognosis of a relapse, as such, is less favorable than that of the first attack, only when there are other undesirable symptoms, or when the patient is pregnant; in the latter class the prognosis is decidedly less favorable than in the non-pregnant.

The prognosis of symptomatic chorea in diseases of the brain and spinal cord is generally doubtful, and in each case depends on the gravity of the original disease.

Treatment.

The treatment of chorea has passed through similar changes to that of epilepsy. From the most energetic technocratic [sic] treatment, with frequent bloodletting and regularly repeated purgations, as recommended by Sydenham, to the most complete therapeutic nihilism, which affirms the uselessness of all remedies on the ground of the theory of the self-limited nature of the disease, we find all stages of transition. The number of remedies recommended and used during the last few centuries is very large.

In general, the treatment of chorea is by no means a thankless task. Without doubt, an intelligent medical action is of decided use, and in many cases has a directly curative effect.

We will first speak of the causal treatment, then consider the dietetic precautions, and finally, the indications and the effects of the most important physical and officinal remedies.

The causal indication cannot in most cases be fulfilled, as the fundamental trouble is usually quite unknown to us, and when understood, it is either such as to be inaccessible to therapeutics, as is the case in local disease of the brain and cord, or else it is only an indirect cause, which gives us no opportunity of exercising a proper curative action upon the chorea, as in anæmia, chlorosis, hysteria, rheumatism, endocarditis. A suitable treatment of these affections may doubtless exercise an alleviating effect upon the symptoms of chorea—although this is not always the case—but it can neither cure nor prevent a relapse. And it scarcely needs to be said that the physician has but little power against the disposition, when created by hereditary influence, bad education, excessive sexual irritation, and self-abuse.

The application of venesection, and local bloodletting along the spine, the application of derivatives, the methodical use of purges—all these have long been given up, partly as useless, partly as injurious. The stools must of course be regulated, if necessary, and intestinal worms be got rid of, but these steps have usually no influence upon the course of the disease.

It is very important to *regulate the entire regimen* with care; not only the bodily, but the mental dietetics. First of all, all occasions for psychical excitement and mental or bodily exertion must be removed. The child must be taken at once from school, and all head-work at home must be stopped. It is very advantageous to spend a long time in the country or on the seacoast. In the case of poor patients, the effect of a well-ordered hospital upon the mental condition, the appetite, and the sleep is very good; they enjoy the quiet of their surroundings, the regularity and order of their daily life, the abundance of fresh air, and they are free from the constant apprehension of meeting with acquaintances. But no more than this need be expected; the duration and course of chorea are seldom affected by a change of place and habits. If self-abuse is practised, the parents must be informed that it is necessary to watch the child. The nourishment should be simple, but abundant and strong; the patient should spend the greater part of the day in the open air, as far as the weather allows.

Sleep is of the very greatest consequence. A quiet and sufficiently long-continued sleep must be secured by avoiding evening excitements, stimulating conversation, and too late meals, by keeping the chamber cool and dark, by preventing noise in the patient's neighborhood, and finally, if necessary, by chloral hydrate. This medicine, in my experience, has proved an extraordinarily valuable remedy in the treatment of chorea, especially in cases where the violence of the jerkings interferes with falling asleep, or the sleep itself is disturbed by spasms. If the intensity of the movements exhausts the patient, I sometimes give another dose during the daytime, in order to procure a few hours of sleep; this usually comes very speedily, and the muscular disturbance is soon quieted.

The amount of bodily exercise proper to be taken should be judged according to the indications of the special case. When the chorea is violent both in the lower extremities and the trunk, walking is not to be thought of; the patient must sit in the open air or be driven.

As to the methodical gymnastics, which some authors recommend as a really curative procedure, and others as an adjuvant, I would postpone it to the period of decline of the disease, and confine the patient to such exercises as expend but a moderate amount of force. The stimulation of the will-power, which is produced by the master's word of command, and the fixing of the attention upon the gymnastic exercises, aid the effect of the latter in ordinary cases, according to Hasse's experience. It need not be said that such efforts must not be made in symptomatic chorea due to a spinal or cerebral disease.

We come finally to the estimate of the *pharmaceutical and physical* remedies recommended for chorea. There are great difficulties in the way of forming a critical judgment of the activity of remedies in a disease whose duration is so variable, whose course is always subject to spontaneous remissions, and which so often passes away quickly and easily without any medication. The statistical method, for these reasons, must be used with the greatest care, and the literary material we possess is not by any means adapted to the formation of statistics. We must therefore test the effects of remedies in each individual case as *objectively* as possible, with careful regard to the individuality of the patient. The disease is so protracted that we have a chance to use one remedy after another, and to compare their effects. This can best be done in those cases which have lasted for three months without improvement. The results of the numerous experiments which I have made in this manner are positive in respect to some remedies and methods, but negative in most.

Of the so-called *nervines*, *arsenic* deserves a most prominent rank. Although it was used in a few cases at the beginning of the present century, it owes its extension to the warm recommendations of Romberg (l. c., p. 533). In spite of this, I am convinced that the admirable effects of arsenic are far from getting the credit they deserve, in text-books and in daily practice.

The dissatisfaction expressed by many authors in regard to the action of this remedy may be traced principally to the smallness of the doses used. I have for many years given, with the best effect, and with no bad consequences, double the doses mentioned in text-books of pharmacy and posology, namely, from five to eight drops of Fowler's arsenical solution for children, and eight to twelve drops for adults, three times a day. Many patients bear these doses for weeks and months without the slightest gastric disturbance; others lose their appetite after using it for a time, and complain of pressure in the pit of the stomach and burning of the conjunctivæ, disturbances which disappear after a few days' suspension of the remedy, and by no means contraindicate a resumption. I have never observed any severe or prominent disturbances.

Let it be remarked, that it is improper to prescribe so powerful a remedy in the form of drops, as the size of the drop is greatly dependent on the form of the edge of the vessel it is poured from.

For this reason I strongly advise to dilute the arsenical solution with a considerable quantity of aromatic water, *e. g.*, cinnamon water, and to administer it from a graduated spoon or vessel.

Steiner, whose observations show that good results can be attained with small doses, associates the arsenical solution with opium when there is much unrest, continuing during sleep; his formula is: Fowler's solution, eight drops; tincture of opium, six drops; water, four fluid ounces. Four tablespoonfuls to be taken daily.

Eulenburg[1] has lately recommended the use of large doses of arsenic, and has proposed *subcutaneous injection* as a means of avoiding gastric disturbances. At the same time, Lewis Smith[2] made a similar recommendation. I have treated a number of cases of tremor, paralysis agitans and chorea with arsenic injections as recommended by Eulenburg, but the painfulness of the injections, and especially the duration of the pain, were so considerable that I had to give them up.

For young people, who, in general, bear pain badly, the subcutaneous injection is not suitable, especially when it is so severely painful as the one spoken of.

The favorable action of arsenic is usually manifest in a week, and two weeks are almost always sufficient—provided that the remedy does not have to be suspended on account of toxic effects—to reduce the symptoms of chorea to a minimum.

In several obstinate cases I have been able to obtain the clearest proofs of the prompt action of the arsenic, after bromide of potassium, oxide of zinc, electricity, and the water treatment had been employed by me with little or no effect. But these were all idiopathic cases. It must be decided by further observations, whether the favorable effect occurs in chorea symptomatic of affections of the brain and spinal cord, and in reflex chorea. The trial is certainly worth making. As regards the other so-called nervines, some have praised the preparation of zinc, especially the oxide, valerianate, and sulphate. So the nitrate of silver, sulphate of copper, and other copper salts, sulphate of aniline, bromide of potassium, calabar bean and its preparations, strychnine, valerian, and asafœtida.

The oxide of zinc was given by Hufeland in doses of fifteen grains three times a day. Steiner combines it with saccharated carbonate of iron in cases of anæmia (saccharated carbonate of iron, thirty grains; oxide of zinc, twenty grains; sugar, two drachms. Make into a powder, of which a knife-pointful is to be taken thrice daily). Sulphate of zinc has recently been praised by Butlin.[3] He begins with small doses and increases to emetic doses, but as soon as nausea is produced, he goes back to smaller doses, or suspends the treatment entirely. Improvement is said always to occur, often cure.

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[1] Zur Therapie des Tremor u. d. Paralysis agitans. Berliner klin. Wochenschrift, 1872. No. 46; and p. 393 of this volume.

[2] Medical Record, 1872.

[3] Lancet, 1871. No. 17 and 18.

The salts of copper are reported by Bergeret and Mayençon[1] as having been used successfully by themselves.

Sulphate of aniline has been given by Turnbull in doses of from three-quarters to one and a quarter grains thrice a day, with reported good success; but Steiner's subsequent experiments gave no satisfactory results.

Bromide of potassium, much recommended, especially by Dumont, Gallard, Hough, Kesteven, has lately been tried carefully by Steiner[2] and myself. Our results entirely agree in this point, that bromide of potassium is not a remedy for chorea; its use, even for several weeks, gives rise to no improvement, or but a trifling amount.

Among the preparations of calabar bean, the powder and the tincture (five parts of the bean to thirty-five of alcohol) are recommended by Harley and Ogle as efficacious in doses of ten or twenty drops three times a day. Eserin, the active principle of calabar bean, has very lately been tried by Bouchut[3] in 437 cases. He gave it pure, sometimes in the form of sulphate, in doses of from one-thirty-second to one-twelfth of a grain (for children from seven to twelve years old), for a considerable period. In doses of one-twelfth of a grain it gave rise to pallor, nausea or vomiting, salivation, sweating; the pupils varied in their behavior; there was a very unpleasant paralytic state of the diaphragm. These troublesome symptoms, which lasted from one to three hours, were never observed by B. after doses of one-twenty-fifth, given twice or three times a day. He states that by this treatment chorea was cured in an average of ten days (?). There have been no control experiments.

The use of strychnia seems to have been entirely given up since the unfavorable criticism of Sée (l. c., p. 502) upon the results of the strychnia experiments made by Rougier,[4] Fouilhoux,[5] and Trousseau.[6] There is hardly any mention made in current literature at present of the use of strychnia in chorea. It is probably best not to resume the use of the medicine.

Hypnotics and anæsthetics may be entirely dispensed with in slight and moderately severe cases; but they are of priceless value in extreme cases, where the excessive muscular restlessness and sleeplessness threaten to exhaust the organism. I decidedly prefer chloral hydrate; it almost always acts promptly and surely; by day or night a dose of from fifteen to eighty grains is followed by quiet sleep and cessation of the muscular activity.

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[1] Journ. de l'Anat. et de Physiol. 1874. 1 seq.

[2] Jahrb. f. Kinderheilkunde. III. 1870. p. 297.

[3] Bulletin génér. de thérapeutique. No. IV. 1875.

[4] Recherches sur la morphine et la strychnine. Lyon, 1843.

[5] Gaz. méd. Paris, Octobre, 1846.

[6] Bull. de l'Académie de Méd. Tom. XII. p. 397. 1846.

Other authors have recommended chloral hydrate in severe cases, as Frerichs,[1] Gairdner,[2] Caruthers,[3] Briess,[4] J. Russel,[5] Bouchut,[6] Verdalle.[7]

Gairdner saw a case of chorea in a girl of eight years, who took by mistake a dose of sixty instead of twenty grains of chloral hydrate, and after recovery from her intoxication was found to have entirely lost her chorea.—Frerichs gave it in the dose of eighty grains to a male of seventeen years with severe chorea. The result was a refreshing sleep of five hours.—Russel obtained good results in the fifth month of pregnancy, after bromide of potassium had been given in large doses without success.—Bouchut's patient of fourteen and a half years, with chorea and dementia, took forty-five grains a day for twenty-seven days, making in all somewhat over two and a half ounces, spent almost the whole time in sleep, and suffered no visible bad results from the chloral. Improvement appeared from the fifth day of the use of choral, and the cure was complete on the twenty-eighth day.

Verdalle's patient of eleven years took very nearly three ounces in fifteen days. The improvement began to appear on the first day the medicine was given, or the fourteenth of the disease.

The fact that this medicine can be safely entrusted to non-medical persons for administration, and the further circumstance that its hypnotic effects are not followed by any bad consequences, give chloral in general the advantage over chloroform.

Chloroform, which was often employed in severe cases before Liebreich's discovery of the therapeutic value of chloral, is praised by most authors of the former time, and by some of the present, as Grossmann, Lowes, Barclay, Banks, Hasse, Ritter. The inhalation, practised once a day or oftener, and carried to the point of half or total narcosis, is preferred to the internal use. Whether choral hydrate is to make chloroform wholly superfluous, is at the present time uncertain, but it may at least be said that the latter will be seldom required.

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[1] Dissertation by *Frick.*

[2] Glasgow Med. Jour. 1870. p. 550.

[3] Lancet. 1870. p. 501. April 2.

[4] Wiener med. Presse. No. 5. p. 102. 1870.

[5] Med. Times and Gaz. Jan. 8, 1870.

[6] Bullet. génér. de thérap. Févr., 1873.

[7] Ibid. Mars, 1873.

The narcotics, especially opium and morphine, are found to act much less favorably than the anæsthetics, and in general may be dispensed with.

Of the physical remedies, mention should be made of electricity and hydro-therapeutics.

Electricity was first used in the form of friction-electricity by de Haën and others; afterwards, in the form of the induced current, it was applied by Duchenne to the nerve-trunks and muscles, and by Becquerel to the skin, it is said with good effect. More recently the galvanic current, from theoretical considerations, has been more employed, and is greatly praised by most electro-therapeutists, as Remak, Benedikt, M. Rosenthal, Onimus, M. Meyer, Seeger, and others. The methods in which it has been employed are very various. Some recommend the galvanization of the sympathetic, others that of the spinal cord, others of the peripheral nerves, with or without the cord; some consider the direction of the current as important, others as indifferent. M. Meyer employed at each session from twenty-four to thirty single shocks of a battery of thirty elements. All praise the quieting effects of the galvanism upon the muscular contractions, and the rapid improvement from one session to the next. I have not had similar success. In four cases, carefully treated with daily applications of a weak current along the spinal column, no improvement worth mentioning occurred.

The *water treatment* has also been urgently recommended by many older and newer authors (Stiebel, Dupuytren, Hasse, M. Rosenthal, and others), but the methods are so various that at present it is impossible to form a proper judgment of their value. All procedures have been recommended, from the protracted warm bath—I refer especially to the prolonged baths at 26° (90° F.), with or without sulphuret of potassium (four ounces to a bath), which have become very popular in France since Baudelocque—to the cold plunge-bath and the cold affusion. Judging from my own experience, I should give the decided preference to the milder procedure, namely, brief rubbing down every morning with wrung-out cloths (one or two minutes, temperature 15° or 16° R. [65° to 68° F. ;]); wet packing, followed by a short, cool shower-bath, and, finally, the latter without packing. This

method is more likely to quiet and refresh the worn-out
han the heroic procedures of the douche, the cold full
ıd half bath. But I will not say that my small observa-
ıs been sufficient to decide these points. The hydriatic
l of treating chorea certainly needs further exact tests.
ɔrevent relapses, the physician should advise a prolonged
ce in the country or the mountains, or on the sea-coast,
of sea-baths or brine-baths, social stimulus, and the care-
oval of all those social and bodily evils which have been
ed at large under etiology as favoring the development of

HYSTERIA.

JOLLY.

HYSTERIA.

Hysteria has been known to, and described in medicine from the most remote period. If one were to enumerate the earlier writings on this subject, almost the whole of early medical literature would have to be cited. We confine ourselves, therefore, to mentioning a few of the more important works of the seventeenth and eighteenth centuries, and then those of modern literature. (Exhaustive quotations from ancient literature are to be found especially in *Dubois'* Histoire philosoph. de l'hypochondrie et de l'hystérie. Paris, 1833.)

Sydenham, Opera medica. Genevæ, 1736.—*Th. Willis*, De morbis convulsivis and Affectionum quæ dicuntur hystericæ et hypochondr. pathologia spasmodica, etc. Op. omnia. T. III. and IV. 1706.—*Stahl*, De hypoch.-hyst. malo. Halæ, 1702.— *F. Hoffmann*, De morb. hyst. vera indole, 1733.—*R. Whytt*, Observations on the nature, causes, and cure, etc. London, 1763.—*Raulin*, Traité des affect. vaporeuses. 1759.—*Pomme*, Traité des affections vapor. des deux sexes. 2. édit. 1765.—*Tissot*, Traité des nerfs et de leur maladies. 1779.

Of our own time, the following of the larger treatises and monographs are to be mentioned: *Louyer Villermay*, Traité des maladies nerveuses ou vapeurs, etc. 1816. — *Georget*, De l'hypochondrie et de l'hystérie. 1824. — *Laycock*, A treatise on the nervous diseases of women. 1840.—*Dubois*, the above mentioned work. — *Landouzy*, Traité complet de l'hystérie. 1846. — *Brachet*, Traité de l'hystérie. 1847.—*Valentiner*, Die Hysterie und ihre Heilung. Erlangen, 1852.—*Briquet*, Traité clinique et thérapeutique de l'hystérie. Paris, 1859.—*Todd*, Clin. lectures on paralysis, cert. diseases of the brain, and other aff. of the nervous system. 1856.—*Skey*, Hysteria, six lectures, etc. London, 1867.—*Amann*, Ueb. d. Einfluss d. weibl. Geschlechtskrankh. auf d. Nervensystem, mit bes. Berücksicht. d. Wesens u. d. Erschein. d. Hysterie. Erlangen, 1874. 9. Aufl. — *Charcot*, Leçons sur les malad. du syst. nerveux. 1872–73.—Further, the chapters on this subject in the manuals and text-books of diseases of the nervous system, of which the following are particularly to be cited in this connection: *J. Frank*, Handb. d. Nervenkr. Uebers. 1843.—*Sandras*, Traité des mal. nerv. 1851.—*Romberg*, Lehrb. d. Nervenkr. 3. Aufl. 1857.—

Hasse, Krankh. d. Nervensystems in Handb. d. spec. Pathol. u. Therapie v. Virchow. 2. Aufl. 1869. — *Eulenburg*, Lehrb. d. functionellen Nervenkr. 1871 (contains no comprehensive description, but much information concerning particular symptoms of the disease).—*Benedikt*, Electrotherapie. 1868.—*Rosenthal*, Handb. d. Nervenkrank. 1870.—*Duchenne*, De l'électrisation localisé. 3. édit. 1872.—*Hammond*, A treatise on dis. of the nervous system. New York, 1872.—Moreover, the text-books and manuals of psychology may be consulted, which nearly all treat more or less exhaustively of hysteria, and also the handbooks of special pathology, especially those of *Canstatt*, *Wunderlich* and others, and the sections referring to our subject in the manuals of gynæcology, of which I would especially mention: *Scanzoni*, Die Krankh. d. weibl. Brüste und Harnwerkzeuge, sowie die dem Weibe eigenth. Nerven- und Geisteskrankheiten. In *Kiwisch v. Rotterau*, Klin. Vorträge u. s. w. 1859.—*Marion Sims*, Clinical notes on uterine surgery. New York, 1865.

Of the numerous papers on hysteria, and particular symptoms of the same, which have appeared in periodicals, brochures, or dissertations, I would cite the following: *Sinogowitz*, Ueber Krampfformen eigenthüml. Art und deren Verhält. zu Sexualstör. bei weibl. Indiv. Rust's Magazin f. d. ges. Heilkunde. 23. Ed. 1827.—*Mondière*, Recherches sur les l'œsophagisme. Arch. génér. 2. Sér. T. I.—*Gendrin*, ibid. 2. Sér. T. XII. (Anæsthésie.)—*Henrot*, De l'anésthésie et de l'hyperesthésie hyst. Thèse de Paris. 1847. — *Beau*, Recherches clin. sur l'anesthésie, etc. Arch. génér. 4. Sér. T. XVI. 1848.—*Macario*, De la paralysie hystér. Annal. méd. psych. 1844.—*Schützenberger*, Recherches clin. sur les causes organ., etc. Gazette méd. de Paris. 1846.—*Szokalsky*, Von der Anästhesie u. Hyperästhesie, etc. Prager Vierteljahrsch. 1851. 4. Bd.—*L. Meyer*, Ueb. acute tödtl. Hysterie. Virchow's Arch. Bd. 9.—*Lasègue*, Sur la toux hystér. Arch. génér. 1854. *Parrot*, Etude sur la sueur de sang, etc. Gaz. hébd. 1859.— *Wunderlich*, Ueb. d. Eigenwärme am Schluss tödtlicher Neurosen. Arch. d. Heilk. 1864.—*Lebreton*, Des différentes variétés de la paralysie hystérique. Thèse de Paris. 1868.—*Bourneville* et *Voulet*, De la contracture hystérique. Paris, 1872.—*Svynos*, Des amblyopies et des amauroses hyst. Thèse de Paris. 1873. —*Ferran*, Du vomissement de sang dans l'hystérie. Paris, 1874.—Then the works on Articular neuroses: *Brodie*, Lectures illustrative of certain local nervous affections. London, 1837.—*Esmarch*, Ueber Gelenkneurosen. Kiel. u. Hadersleben, 1872.— *Wernher*, Ueber nervöse Coxalgie. Deutsche Zeitschr. f. Chirurgie. Bd. I.—*Stromeyer*, Erfahr. über Localneurosen. Hannover, 1873.—*Berger*, Zur Lehre von d. Gelenkneuralgien. Berl. klin. Wochenschr. 1873.—Also of the numerous treatises on spinal irritation: *Stilling*, Physiol. patholog., etc. Unters. üb. die Spinalirritation. Leipzig, 1840.—*Hirsch*, Beiträge z. Erkenntniss u. Heilung der Spinalneurosen. Königsberg, 1843.—*Tuerck*, Abhandl. üb. Spinalirritation. Wien, 1843.—*Mayer*, Ueber die Unzulässigkeit der Spinalirritation. Mainz, 1849, and others.

General Observations.

The multiform symptoms which are included under the term hysteria may, without exception, be recognized as the consequences of a functional disturbance of the nervous system. The disease therefore belongs to the class of neuroses, and to those neuroses to which, in the absence of a sufficient knowledge of their anatomical basis, the term functional must still be applied. Hysteria is further to be described as a general neurosis, for its symptoms allow one to infer a participation of the most various portions of the nervous system—certainly of the brain and spinal cord, and, with much probability, of the peripheral, and particularly also of the sympathetic nervous system.

The most constant symptoms of hysteria are those which show a condition of increased irritability to physical and psychical stimuli. A peculiar psychical behavior is partly an immediate consequence of this abnormal irritability, and is in part the expression of greater disturbances of brain power. The function also of motor portions of the nervous system is, as a rule, upset, partly because these receive an increased impulse on account of the exaggeration of sensory irritability, both in a reflex and in a psychical manner, and partly in consequence of a direct exaltation of their own irritability.

Pain, neuralgia, and hallucinations in the sensory, and convulsions in the motor tract are the further consequences of increased irritability.

But there occur in hysteria, not only the symptoms of increased, but also those of diminished irritability, both in the sensory and motor sections. Anæsthesia may take the place of hyperæsthesia, paralysis that of convulsion, and these opposite conditions frequently exist simultaneously in different portions of the nervous system.

Finally, there occur, as symptoms of an apparently altogether different nature, those of altered power of the organs of secretion and excretion. Yet these, too, may undoubtedly be recognized as dependent upon an influence of the nervous system.

The authority for including all these various symptoms in one uniform class of disease is derived from the frequency of their simultaneous occurrence, and from the circumstance that they may appear and vanish alternately, and that, seem they never so obstinate, and apparently quite localized in any given section of the nervous system, yet, ultimately, they can always be proved to depend upon the general condition of the latter.

The influences under which this condition develops itself are various. Sometimes there is a peculiar reaction (*Reaction*) implanted in the nervous system from birth; at others it is caused by an abnormal quality of the nutritive fluid; again it is the consequence of irritants which operate, through the sensory nerves, on the central organs; or, finally, it results from so-called psychical stimuli.

The circumstance that hysteria occurs more frequently in the female than in the male, and that different physiological and pathological processes in the female genital organs have an unmistakable influence in its production, has given rise to the erroneous assumption that hysteria is a disease exclusively peculiar to the female sex, and that in such it invariably proceeds from disease of the genital organs (hence also the name, no longer to be got rid of, from ʽυστέρα, the uterus). Unmistakable as is this connection in many cases, it merely indicates one of the various ways by which similar functional disturbances of the nervous system may be caused.

Etiology.

Only in the minority of cases of hysteria can those causes which develop the predisposition to the disease be separated from those which cause its actual outbreak. Frequently both occur together, or that which in one case stands in a causal relation may in another produce the predisposition. A separate enumeration of both series of cases would therefore only lead to tedious repetition, while a consideration of the one must also show its varying import in two directions.

We commence with the consideration of the *differing liability of the two sexes* to hysteria. That the latter is a disease occurring with preponderating frequency in *the female* has already been pointed out, as also that it is not exclusively peculiar to her. In fact, all the characteristic appearances of hysteria may occasionally be observed in men—the psychical condition as well as the sensory and motor disturbances. Nevertheless, it is difficult to supply exact information regarding the relative frequency of this occurrence, as there exists, in general, a tendency to restrict as much the diagnosis of hysteria in men as it is usual to extend it in women, and as, in consequence of this tendency, hysteria is very often designated hypochondriasis in men, and hypochondriasis hysteria in women. One can therefore only ascribe to the statistics which have been drawn up regarding the different sexual tendency to hysteria the significance of a general approximation to the actual state of affairs.

Thus, Briquet states that, of 1,000 cases of hysteria which he in part observed himself, and in part collected from the statements of others, 50 occurred in men. So also he found the ratio among the predecessors of the hysterical persons he observed, viz., 204 hysterical women to 11 hysterical men. Thus one would have to assume a tendency to the disease twenty times greater in the female than in the male.

We can only at present conjecture what are the real causes of this unequal tendency. In each case we must distinguish two modes of its production, viz.: there may be, in the first place, *congenital peculiarities* which render the nervous system of the female especially liable to a development of the disease; and, secondly, the blame may be imputed to *acquired conditions*, partly due to the physical function peculiar to women, and partly to their social position.

With reference to the first point, a congenital difference of predisposition in the two sexes flows immediately from the fact that even in childhood hysteria affects girls much more frequently than boys. Here, at all events, there can as yet be no question of a difference of physical function. The significance of the latter, however, shows itself undoubtedly in more advanced life, when we see how frequently diseased and excited conditions of the female genital organs occasion hysteria, and how a tendency to the same diminishes at an age when the function of those organs ceases.

The *frequency* of the appearance of hysteria is very different at different ages. In childhood, long before puberty, well-marked hysterical phenomena occur. Briquet states that in so much as one-fifth of all cases the development of the disease takes place before the twelfth year. Although the correctness of this statement has been much doubted, it appears to me from my own observations to approach tolerably near the truth. On the other hand, the development of hysteria at an advanced age, after the close of the climacteric period, is rare. It undoubtedly arises most frequently at puberty, and during the following decade.

The following statistics on this point may here be given: of 268 cases, observed by Amann, 16 occurred at an age between eight and fifteen years; 62 between fifteen and twenty-five; 92 between twenty-five and thirty-five; 81 between thirty-five and forty-five; 12 between forty-five and fifty-five; and 5 between fifty-five and seventy. Moreover, in the following table, the results of the researches of Landouzy, Briquet and Scanzoni are placed together:

Outbreak of hysteria at the age of	Number of cases of			Total.
	Landouzy.	Briquet.	Scanzoni.	
0–10 years..........	—	66	—	66
10–15 "	48	98	4	150
15–20 "	105	140	13	258
20–25 "	80	71	64	279
25–30 "	40	24		
30–35 "	38	9	78	49
35–40 "	15	9		
40–45 "	7	1	44	63
45–50 "	8	3		
50–55 "	4	3	11	24
55–60 "	4	2		
60–80 "	2	—	3	5
	351	426	217	994

The harmony of the above figures is by no means sufficient for furnishing absolutely certain results with reference to the frequency of the appearance of hysteria at puberty, and during the succeeding term of life, till the commencement of the climacteric period. Briquet considers the period of puberty as by far the most favorable, and in support of this advances the fact that of his 426 cases, 221—that is, more than half—developed themselves between the twelfth and twentieth years of life. The figures also of Landouzy agree with this, while, on the other hand, according to the statistics of Scanzoni and Amann, the disease occurred most frequently in the third and fourth decades. One must, however, remember that both the latter statistics are the result of gynæcological practice, and that an unusually large number, at all events, of these cases of hysteria occurred in connection with disease of the genital organs; and further, that these latter forms develop themselves most frequently during that period in which the genital function is most active, and thus one may regard the results attained as less generally applicable, and rather as deciding in favor of Briquet. Testimony is, however, agreed upon this point, that in the fifth decade the development of hysteria is much rarer than in both the preceding decades, and that in the succeeding period a much more marked diminution takes place.

The climacteric period itself is, as a rule, considered favorable to the production of hysteria, and this view receives the greater probability from the fact that the various psychical diseases nearly related to hysteria develop themselves with comparative frequency at this time. From the statistics given, however, no support is gained for this view.

Among the most important causes is a *hereditary liability*, which may produce a tendency to hysteria, and a complete development of the disease. With especial frequency does an inheritance of like tendencies (gleichartige Vererbung—Hérédité similaire of Morel) exist here, so that the hysteria of parents or forebears generally has as a consequence hysteria of the offspring. Particularly does the transmission of the disease from mother to daughter occur with great frequency. But, moreover, the various other nervous disorders, which have appeared in the forebears, favor a disposition to hysteria in the descendants, just as it happens, on the other hand, that hysteria appears in one generation, and epilepsy, chorea, insanity in the next. These relations may, from direct evidence, be recognized in numerous individual cases. The study of mental disturbances, in particular, has furnished a mass of such facts. But they are scarcely yet to be expressed in definite figures, as too great difficulties underlie the exact arrangement of all the peculiarities of a sufficiently large number of individual cases. Here again, on account of the importance of the matter, the statements of Briquet may be reproduced, but it must, at the same time, be doubted whether they are generally applicable.

Briquet obtained particulars concerning the parents, brothers and sisters of 351 hysterical persons, and found that of these (in all numbering 1,103) 214 suffered from hysteria and 58 from other diseases of the nervous system. Thus in all, nervous diseases were proved to exist in about 25 per cent. of the nearest relatives, 167 non-hysterical persons, whose family history was in like manner examined, had among 704 of their nearest relations (parents, brothers, and sisters) only 11 who suffered from hysteria, and 4 from other diseases of the nervous system—that is, rather more than 2 per cent.

As regards the different influence of fathers and mothers, Briquet found that of 282 fathers of hysterical patients of whom he could obtain accounts 6 had suffered from hysteria and 20 from other nervous diseases, while of 317 mothers there were 103 cases of hysteria and 6 of other diseases of the nervous system. Further, there were many more who suffered from hysteria and other nervous disorders among the sisters than among the brothers of the hysterical.

Moreover, Briquet proved the following with respect to the female descendants of the hysterical: Of hysterical women who bear daughters, rather more than the half transmit the disease to one or several of these, and again, rather more than a half of the daughters of the latter (*i. e.*, granddaughters) also become hysterical. In all, then, rather more than a fourth part of the female descendants of the hysterical suffer in their turn from hysteria.

A conclusion cannot be drawn from Briquet's statements as to the point, among how many of his patients generally, inherited conditions of any kind whatsoever existed, but it is certain that the number of these must have been very large. Amann has recently stated that in 208 cases of hysteria he proved with certainty a hereditary tendency 165 times—that is, in 76 per cent. But here there lacks any more exact information concerning the nature of the conditions originating the transmission.

Difficult to state, but certainly of decided influence upon the transmission of a tendency to hysteria, are such peculiarities of the parents as do not yet come under a definite class of disease, but which nevertheless indicate an abnormal condition of the nervous system: unusual irritability and sensitiveness, a tendency to excited behavior (explosives Handeln), and an increased participation of the nervous system on the occasion of various bodily ailments;—in short, all those phenomena which one is wont to describe collectively as the expression of a neuropathic disposition or nervous temperament, and which may go on to hysteria and other diseases of the nervous system.

Moreover, various weakly conditions in the parents, arising from chronic diseases or otherwise, seem to favor the development of a hysterical tendency in children. To phthisis especially is such an influence ascribed. Privation, insufficient nourishment, and further, an advanced age of the parents at the time of birth operate similarly.

The transmitted tendency to hysteria may for long remain latent; in many cases it requires the addition of a further cause to effect an outbreak of the disease; in others, again, the congenital taint is sufficiently strong to develop it independently, sometimes at so early a period of childhood that it is scarcely possible to discover a time when absolutely no symptoms of the disease existed.

It is feasible to suppose that, with so singular an organization of the nervous system, peculiarities of physical construction are connected, from the presence of which a disposition to hysteria may *à priori* be recognized, as also that such peculiarities generally, without being inherited, may give rise to a special proneness to hysteria. Yet, hitherto little that is satisfactory has been discovered upon this point. One meets with the hysterical tendency in constitutions the most various; robust, full-blooded individuals as well as the weakly and poorly nourished may be affected with it, and also those of a perfectly healthy constitution. Nevertheless the *greater predisposition of weakly constitutions* is not to be denied, just as we shall also find that acquired conditions of debility favor the development of the disease. Such a condition also probably forms the connecting link in those cases in which debility of the parent induces hysteria in the descendants.

Certain peculiarities of the *psychical constitution* favor more than others the development of hysteria. Particularly such individuals as combine with a lively disposition and a tendency to powerful and changing emotions little strength of will, are prone to hysterical ailments, as it is also just these psychical peculiarities which appear in a pronounced manner in the picture of complete hysteria. All circumstances which favor such a psychical condition also favor the development of hysteria. On the other hand, the degree of intelligence appears to be quite immaterial in this relation. The disease is observed to develop itself equally in persons highly gifted mentally, in those of less capacity, and, finally, in the actually imbecile.

If we now pass to a closer consideration of the *physical influences* which promote a development of the hysterical tendency, and which can mature an existing predisposition, we find in the first rank such causes operating as result in a permanent and general weakening of the organism. *Primary anæmia* progresses almost, as a rule, to hysterical symptoms; but one does not rarely observe *secondary anæmic conditions*, which develop themselves after great losses of blood, after exhausting diseases, or in consequence of insufficient nourishment, also giving rise to hysteria. This is seen, for example, during convalescence from severe attacks of fever, and in the course of phthisis, but with especial frequency in chronic affections of the stomach and abdomen. Of the latter, it is particularly *diseases of the female genital organs* to which a distinguished part in the causation of hysteria has at all times correctly been allotted.

The influence of these diseases makes itself apparent in many ways, partly thus: that the chronic derangements in the nutrition of the entire organism, the general anæmia, produce an abnormal state of the nervous system. There is, in addition, however, a specific influence. The peculiar nervous phenomena which appear in otherwise healthy women at the period of menstruation, during gestation, and in consequence of the excitement connected therewith, offer a proof that these organs possess an especial relation to the nervous system. This shows itself none the less plainly by the fact that in many cases of pronounced hysteria decided exacerbations of existing symptoms occur during menstruation and gestation. Most clearly, however, does this appear in those cases in which symptoms of hysteria are called forth by irritative pathological conditions in the genitals, to alter in intensity with the variations in these conditions, and to vanish with their cessation. In all such cases the influence of the central nervous system is unmistakable, and in great part induced by the centripetal nerves of the sexual organs which are excited by pressure, irritation, or inflammatory processes. As a rule, moreover, the circulatory apparatus is directly implicated at the same time, as sometimes, on account of the local congestion, and the losses of blood in consequence thereof, anæmia is induced in other parts, and, again, owing to a failure on the part of the vascular system to empty itself normally, plethora and congestive phenomena are produced. If, on the whole, more rarely than anæmic conditions, nevertheless these latter states of *abnormal vascularity* most undeniably sometimes help to develop the hysterical tendency. In these rarer cases one observes the symptoms of the disease disappear if an abundant evacuation of the blood vascular system takes place. One must, however, be cautious not to deduce a condition of generally increased vascular fulness from attacks of "rush of blood to the head," which appear as one of the phenomena of hysteria, but also occur in anæmic subjects.

Great, however, as is the significance in hysteria of physiological and pathological processes in the female genital organs, it must nevertheless be distinctly stated that it may develop itself quite independently of their influence. Not only does the occurrence of hysteria in men prove this, but also its appearance in women at a time of life when the genitals play no part; and, further, the circumstance that one meets with it in mature life without any trace of an abnormal condition of the genital organs.

Scanzoni and Amann found among the hysterical patients, whom they examined, nineteen to twenty per cent. whose genitals were quite healthy. The statements of von Franque give a similar result. As in the practice of these three gynæcologists, at any rate, cases of hysteria with disease of the genital organs occur with unusual frequency, one may, according to these statements, say that at least a fifth and probably a much greater proportion of all hysterical women are free from such diseases. From a collection which Landouzy made of the reports of autopsies, from ancient and modern literature, it appears that in forty cases of hysteria, thirteen times, that is, in a third, changes in the genital organs, whether the uterus, the ovaries, or the Fallopian tubes, were absent. It must, however, be determined by much more extensive statistics whether these numbers express the true ratio. I myself believe from personal experience, though I cannot give figures, that on investigation the presence of disease of the genital organs will be proved in scarcely a half of all hysterical cases. It must, moreover, be emphatically stated that even when they are present, these diseases frequently play the part of an exciting cause to a pre-existing tendency to hysteria; and, finally, that they may also make their appearance as quite accidental complications. Conversely, as regards the frequency of the occurrence of hysteria with affections of the genital organs, the statements of different gynecologists on this point vary so extremely one from another that we must, in the meantime, refrain from drawing any conclusion on this head. Statements regarding the significance of different kinds of disease of the sexual organs are more agreed. Thus, it is generally stated that it is the most serious disorganizations of the genitals (cancer of the uterus, etc.) which furnish the smallest percentage of hysterical patients, while of women affected with versions and flexions of the womb, as well as with chronic inflammatory conditions of that organ and the ovaries, a comparatively large number suffer from hysteria. This is also not rarely observed in persons with an imperfectly developed uterus, and one sees it likewise in those whose uterus is altogether wanting.

Of especial significance in any case are the processes in the genitals during *gestation, delivery, and lying-in*, both from the direct influence which they exercise upon the organism, and from the numerous diseases of the sexual organs which follow in their train. Scanzoni states that, of 217 hysterical patients whom he had treated, 165, or 75 per cent., had been puerperal, and that, of the latter, not less than 65 per cent. had borne children more than three times.

Menstrual disturbances, even without disease of the genital organs, occur in many hysterical women, without its being always possible to state in how far they precede or cause the disease as an independent functional disturbance, or whether they are merely to be regarded as a symptom and consequence of it. At any rate, long-continued metrorrhagias may give rise to hysteria by means of the exhausted conditions which they bring about. But, as above stated, the increased vascular fullness and irregularities of circulation, caused by a suppression of the menses, can likewise influence the nervous system in a similar manner.

That hysteria cannot, as formerly believed, be exclusively "virginum et viduarum affectio," is satisfactorily proved by what has been said. But there are undoubtedly cases of hysterior in which the *non-gratification of the sexual appetite*, *i. e.*, sexual abstinence, exercises a certain influence as an exciting cause. Especially in the case of young widows, who were formerly in the full enjoyment of sexual gratification, and in those women who, from impotence on the part of their husbands, cannot obtain the said gratification, does the disease sometimes develop itself, and may be cured under circumstances which remove the cause.

More frequently, however, than sexual abstinence does *sexual over-irritation*, particularly that induced by onanism, cause the disease. It is also the latter agency which is found with comparative frequency to be causal in cases of hysteria in men. Moreover, in them chronic diseases of the urethra and prostate seem to play a part. Yet there are no corresponding statements extant with reference to the frequency of these relations (in man), as there are with regard to those analogous in the female.

The last-mentioned causes of hysteria are in no case to be regarded as operating more than in a purely physical manner; but herewith there is always observed a simultaneous influence acting in a directly psychical manner. Psychical influences generally, operating in part independently and partly in conjunction with physical causes, have the most important bearing upon the development of the hysterical condition. On this point, it may be said, as a rule, that persistent mental emotions, especially those of a depressing nature, operate in this way. The sad sense of failure in one of the objects of life, which so often, in particular, affects childless women and old maids; in other cases, love-sickness and jealousy; in others, the feeling of injured vanity and wounded pride; in others, self-reproach because of secret sins; and, finally, grief and anxiety on account of all the possible accidents and relations of life, are a few of the more important emotions in question, which need not be further particularized. The effect of these varies extremely, according to the previous condition of the psychical soil. The more sentimentality there exists, and the less the individual is accustomed to repress his feelings, the more easily do these assume a pathological character and influence the whole state of the nervous system. There are, however, psychical excitations so powerful as to overcome an otherwise perfectly healthy constitution. Thus, one sometimes observes hysteria suddenly appear in persons hitherto healthy, after very violent excitement from fright and fear, or after very acute mortifications and injuries of one's sense of honor, especially after rape. Much more frequently, however, all these influences are but the exciting causes of an outbreak of the disease (already existing) or of some of its paroxysms.

From all that has been said, it is clear how great an influence *education* may have both upon the repression and upon the development of the disease. By means of appropriate physical and moral training, the predisposition to disease may be extinguished, just as, conversely, the tendency, existing only in a slight degree, or not at all, may be artificially roused. All influences which favor physical debility are active in the latter direction; that is, both a too delicate nurture, by which a necessary degree of robustness fails to be imparted to the body, and a too Spartan rearing, which exacts efforts beyond the power to perform them. Both extremes are also pernicious in a moral sense. By too great indulgence, pusillanimity and peevishness are fostered, which prepare the way for hysterical phenomena; by undue rigor and intimidation, such violent disturbance of the feelings may be called forth, that exalted irritability of the entire nervous system, coupled with indecision of character, is the result, calculated to favor the disease. An unsettled, crotchety education, however, now exceeding in one direction and again in another, is fraught with the greatest danger. In schools, and particularly in girls' boarding-schools, the foundation of hysteria is likewise often laid. Especially dangerous are too great demands upon the mental capacity, overburdening with lessons, and at the same time stimulating an over-driven ambition. The want of sufficient physical exercise also follows this as a second pernicious element, and frequently enough it is just by this injudicious manner of life that the inclination to masturbation is awakened.

The influence of a faulty education in the development of hysteria cannot be fully estimated, if one does not take into account a co-operating circumstance, which we find even at a more mature age as a causal element of hysteria, viz., the rise of *an imitative impulse.* Hysterical mothers transmit not only the seeds of disease to their children; they also favor its development by education and by their own example. Above all, it is those symptoms which occur in paroxysms, especially convulsions, which provoke imitation. But the whole mode of feeling and thought also transfers itself from continued intercourse. As in children, so also sometimes in nurses who have for a length of time attended hysterical patients, this so-called imitative infection is operative; or in other patients who have been nursed beside such; and, above all, in people who have been the accidental witnesses of an hysterical attack. In this way an actual epidemic of hysteria may take place, as is shown by the convulsive epidemics of former ages, and the less extensive attacks of the same nature which occur in hospitals, cloisters, factories, girls' schools, etc., at the present day.

Moreover, one usually finds this sort of infection only operative in such individuals as are already otherwise disposed to hysteria, and the epidemic spread of the disease is usually ushered in by general predisposing influences. Thus, as a rule, want and misery, in consequence of war or the ravages of disease, or owing to the failure of crops, and famine, have cleared the way for those convulsive epidemics which have appeared and spread through whole districts and populations, usually in connection with great political and religious disturbances. In the limited epidemics in hospitals, debility resulting from other diseases, as well as the enforced inactivity, are to be taken into account as co-operative elements. In educational institutions it is the defects already mentioned in the physical and psychical régime which may induce the tendency simultaneously in a larg number of persons.

Attempts to establish a special tendency to hysteria in certain classes of the population, especially in certain ranks and callings, have as yet led to no positive results, although without doubt a difference of disposition must arise from a variety of external circumstances. A definite influence of nationality and climate has also as yet failed to be proven.

Pathological Anatomy and Pathogenesis.

One can only speak of changes in the central nervous system in a negative sense. Though we discover in a few exceptional cases grosser lesions of the brain and spinal cord of various kinds, yet these can only, in the most favorable cases, be regarded as a few of many influences under which like functional disturbances occur. In other cases, again, it is certainly only a question of accidental complications, as palpable diseases of the nervous system develop themselves in individuals, who are hysterical independently of such. The further possibility of a gradual development, from long-continued hysteria, of structural changes, in portions of the nervous system which at first were only abnormally active, requires more proof, and as yet facts are wanting.

The cases of Charcot, in which after long-continued "hysterical" contractions, sclerosis of the lateral columns of the spinal cord was found, appear to argue in favor of this view, and it is not to be denied that here it was not merely a question of complication in the afore-indicated sense. Even in cases of acute fatal hysteria, changes in the central nervous system have failed to be discovered.

That some alterations in the nature of the nervous system must be at the foundation of its altered function cannot be doubted. But whether we are to expect that those changes affect the structure of the nerve elements, or only have reference to its chemical and molecular constitution, cannot yet be stated. It is quite as possible that the microscope may at some time give us information concerning the nature of this condition as it is that no information may be derived from this source.

One can therefore, in the meantime, only discover the basis of hysteria in an "abnormal" condition of the nervous system, which can only be determined by its symptoms. But this condition, from what we know of the etiology of the disease, may be one either implanted in the nervous system from birth, or acquired partly by sensory irritation and partly from the operation of an abnormal quality of blood. It develops itself most powerfully when all these influences combine.

All theories of hysteria which attribute its origin exclusively to the genital organs, are fallacious, whether it be that the connection is explained as reflex from, or due to changes in the blood of those organs. These causes, however, may well play a considerable part in the production of particular symptoms.

In the oldest theories of hysteria the uterus exclusively acted as a "primum movens" to the disease. The ancient Greek physicians gave it credit for wandering through the whole body, and producing by pressure on different organs symptoms of disease in the latter. When Galen demonstrated the impossibility of such migrations, the opinion arose that, by the retention of seminal fluid or blood in the uterus, a pernicious influence was exerted upon the whole organism. Sometimes this was effected by a decomposition of the fluids, sometimes by the invasion of hurtful gases (Vapores), and again, by pressure of the distended uterus upon the surrounding nerve distribution (per consensum). Later, this explanation was generalized so that irritative conditions of the sexual organs, as a whole, were supposed to be at the foundation of hysteria—a view which was last defined by Romberg thus: hysteria is a reflex neurosis caused by genital irritation. All these theories contain a particle of truth, as they are founded upon a knowledge of particular and frequently operative causes of hysteria. That they, however, do not suffice follows from all that has been stated. The other view, that the disease has its root in the nervous system, and may be developed in this by the most varied external influences, has, since the time of Sydenham, gained more and more adherents, and may at present be said to be that which is almost universally entertained. Opinions concerning the essence of the disease have truly been diverse. Sydenham considered an ataxy of the emotions in the brain to be its basis. Pomme thought it was caused by a hardening (Racornissement) of the nerves. It cannot be said that the ideas we now form of a "peculiar constitution of the nerve-elements" are much clearer than those connected with the expressions quoted, when they first came into use.

Finally, the question is, In what portions of the nervous system have we to look for the *seat of the individual symptoms* of hysteria?

We meet with undoubted *brain symptoms*, not only in great disorders of consciousness during hysterical attacks, but much more commonly and more constantly in the almost never absent (though, of course, present in very varying degree) exaltations of psychical irritability.

This finds expression not only in the subjectively felt, powerful excitement of emotion on little provocation, but also in a series of convulsive movements perceptible objectively, and taking place involuntarily. The following are merely exaggerations of the motor symptoms accompanying the affection even in healthy people, viz.: alterations in the heart's action and in the respiratory movements, spasmodic contractions in the digestive canal, cramps in the most widely differing portions of the external muscular system.

The same phenomena may also in such cases be called forth by means of purely sensory stimulation.

Here, then, we have to do with *diseased reflex actions* of different kinds, whose reflection takes place in the brain, and whose cause is conjecturally to be sought for in *a condition of exalted irritability in the sensory portions* of that organ. The hallucinations occurring in many cases also prove the existence of such a condition. Further, we find a still greater participation of the brain, in states of more complicated mental disturbance, taking place in the hysterical.

We have now, however, to consider whether all the symptoms of hyperæsthesia, pain and spasm occurring in hysteria, are to be referred to an affection of the sensory portions of the brain. This may, indeed, be possible, but it does not appear altogether probable. Against it particularly does the circumstance speak, that frequently in the hysterical, by stimulation of the circumscribed hyperæsthetic and painful spots, strong local reflex actions are given rise to in their neighborhood, and that, then, by a continuation of the stimulus, a gradual spread of the movements follows, in a manner exactly resembling the theory of reflex actions in the spinal cord. It is therefore probable that some hysterical hyperæsthesias and reflex spasms are to be attributed to a condition of *increased irritability in the sensory portions of the spinal cord.* This condition may extend over the whole length of the spinal cord, or exists only in some parts of it.

The pains and hyperæsthesias, however, occurring in particular vertebræ in many hysterical people, have erroneously been produced as the proof of such an irritative condition—the so-called spinal irritation.[1] Vertebral pains may be absent, even when the signs of increased irritability mentioned are distinctly present. The tender vertebræ also do not by any means always correspond to those portions of the spinal cord which, from other symptoms, we must consider to be specially irritable. The painful vertebra frequently alters its position without being accompanied by other changes. In fine, it may, as well as other pains, owe its origin to a purely peripheral irritation.

Lastly, it is possible that in the hysterical there also occurs an *exalted irritability of the peripheral sensory nerves*, which is accompanied by symptoms quite similar to those of increased irritability of the spinal cord, viz., by pain, hyperæsthesia and reflex spasms. Direct proof, however, of affections of this kind does not exist. But more than once already fatal errors have been committed in this respect, in the case of hysterical patients, as circumscribed and very obstinate pains in peripheral portions of the body, especially in the extremities, have been considered evidence of peripheral disease, and it has been attempted to remove them by amputation of the part. The unaltered continuance of such pains, even after repeated amputation (in one case

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[1] Under the term spinal irritation have been, and indeed still are classed, all possible symptoms which one believes may be referred to an irritative condition of the spinal cord. Hence, sometimes vertebral pain is regarded as a symptom pathognomonic, and in itself proving the presence of that condition (by some, indeed, pain in the vertebræ is called spinal irritation), and again other conditions, the most widely differing, pains and hyperæsthesiæ, convulsions and paralyses, even psychical symptoms of all kinds, are regarded as evidences of spinal irritation, so that the latter constitutes in a great measure the clinical picture of hysteria, and, in part, also that of hypochondriasis. The irritation itself is partly looked upon as a purely nervous condition, and is, in part, referred to hyperæmia of the spinal cord. *Hammond* has recently expressed the belief that it may be explained by anæmia—and that, too, a special anæmia of the posterior columns. There is, however, as little proof for the one view as for the other.

Mayo on this account undertook exarticulation at the hip-joint) has afforded the certain proof of its having been merely a case of the excentric transmission of central irritative conditions.

Nevertheless, it must not be considered impossible that peripheral nerves may also, in cases of hysteria, be brought into a state of altered sensibility.

That which applies to hyperæsthesias, is also applicable to anæsthesias. The impediment to conduction may be situated at the most various portions of the sensory tract. It is *à priori* probable, that cases of well-marked unilateral anæsthesia are caused by changes in the brain itself. On the other hand, the circumstance that (even in cases of unilateral anæsthesia) the anæsthetic parts occasionally lose at the same time reflex irritability, only points to the fact that in the peripheral portion of the tract likewise, there exists an impediment to conduction, either in the spinal cord or in the sensory nerves themselves.

The motor may in part be considered a direct consequence of the sensory irritative phenomena. The brain as well as the spinal cord claims the agency. But it is extremely probable that a portion of the spasms which occur, result from a direct increase of the motor irritability without such a sensory excitation. The proof, at least, of a sensory stimulus standing in a causal relation is not often obtained. These spasms may, however, arise, sometimes in the brain and sometimes in the spinal cord. Nor, finally, is the opinion unjustifiable, that the increase of reflex irritability is in some cases to be ascribed to the suspension of the normal reflex inhibition proceeding from the brain.

Lastly, hysterical paralyses are not to be localized with absolute certainty. That hemiplegias proceed from the brain, and paraplegias from the spinal cord, is argued for by the analogy with cases in which there is anatomical change in these parts. It is, however, just as easy to imagine that the converse is true, and most probable that both occur. That the impediment to conduction may be situated in the peripheral motor nerves is improbable from the fact, that, even after the long-continued existence of such paralyses, no changes in the electrical irritability arise. Some hysterical paralyses again seem to belong to the category of reflex paralyses. Hysterical contraction, it has already been mentioned, is regarded by Charcot as the evidence of an affection of the lateral columns of the spinal cord.

Whether we are to regard changes in function of the vasomotor and secretory nerves, which play a distinguished part in many cases of hysteria, merely as dependent upon the central disturbances, or whether peripheral alterations of function also occur in such cases, is not decided; the latter, however, is not improbable.

Symptoms.

General Description of the Disease.

The symptoms of hysteria develop themselves in the majority of cases so gradually, that it is difficult to predict a definite result. The first symptoms are generally those of increased psychical irritability and peevishness, with which hyperæsthesias and conditions of excitement in the region of different senses and exaggerated reflex phenomena are associated. In other cases the latter changes take place first, and the psychical alteration shows itself later. Frequently many years pass before others are added to these symptoms; but often also anæsthesia and paralysis develop themselves early, in particular parts of the body.

There is another series of cases, in which the disease seems to begin suddenly, and to be accompanied, too, by one of those fits which so often appear in the further course of hysteria. Closer investigation shows that in the majority of these cases, prodromic symptoms have already occurred for a longer or shorter period, and that the fit merely signifies a rapid culmination of certain groups of symptoms. As an appreciable external cause is frequently at the bottom of the attack, and as thereafter the collective hysterical symptoms are as a rule more distinctly pronounced than formerly, the mistaken idea easily arises that the whole disease has commenced suddenly.

In a smaller number of cases, however, it does actually develop itself suddenly, or at least within a few days or weeks, in people previously quite healthy. These are cases in which it is induced by very violent moral shocks, or great physical exhaustion (loss of blood, severe febrile diseases, and so on).

In their further course the attacks referred to, which consist partly of more or less general convulsions, and partly of peculiar psychical disturbances, may play so prominent a rôle as to appear to constitute the entire clinical picture, so that, on superficial observation, all symptoms of disease may seem to be absent in the intervals between them. In other cases, after the attacks, widespread paralyses, contractions, anæsthesias, and so on, develop themselves, or those already existing are aggravated. In yet other cases, the attacks are less violent, and separated by long intervals, and it is just in these intervals that severe hysterical symptoms develop themselves, compared with which the attacks are of less importance. Finally, there are numerous cases of hysteria in which distinct fits are altogether absent.

The various symptoms of hysteria are developed, as these short statements indicate, in the most confused alternation, simultaneously and in succession. It is, therefore, impossible to adhere in the description to the order of their appearance; one can only arbitrarily enumerate them one after another, and so point out, as far as possible, the frequency of particular symptoms and their mutual relations. Sometimes all these symptoms are observed in a single case, and thus produce an extremely varied and multiform clinical picture. In other cases only some of them are present, but these are of so much the greater obstinacy and persistence. It will not, however, be possible to maintain, in accordance with these differences, the existence of different forms of hysteria, distinguished by the predominance of one or other symptom, as by means of the various transitions which occur, the connection of individual cases will be proved. Never absent are the peculiar alterations of psychical functions, to which particular disturbances of sensibility and motion stand in the closest relation. Around these the remaining symptoms group themselves, according to the intensity and general prevalence of the disease.

Description of Particular Symptoms.

Disorders of Sensibility.

We commence with the phenomena of *increased sensible irritability.*

Hyperæsthesia, in one form or another, is never absent in the course of hysteria. General hyperæsthesia is met with, affecting all the senses, as well as that limited to the region of particular senses or portions of the same. But, as in other diseased condi tions, so in hysteria, that form of hysteria which consists in an especial acuteness of sensory perception is less frequently met with (increase of differentiating sensibility [Unterschiedsempfindlichkeit], diminution of reflex contraction [Reizschwelle]). As a rule that form is most frequently present (also called hyperalgia and hyperalgesia), which is distinguished by an aggravation of the various kinds of pleasure and aversion called forth by sensory stimuli. If this condition exists in greater intensity, pleasure disappears altogether, and stimuli generally perceptible by the senses produce discomfort and pain.

Not rarely such a degree of hyperæsthesia is only produced by stimuli of a particular kind. There are some sensations, to others indifferent or pleasant, which in the hysterical produce the highest degree of discomfort (Idiosyncrasie); so also there are particular stimuli which are to others a matter of indifference, or unpleasant, and which awaken pleasure in the hysterical and are by them eagerly sought after (Pica).

Finally, with the hyperæsthesia to sensory impressions there is always coupled, in greater or less degree, a similar condition towards psychical emotions. Such also as have no connection with previous conceptions are accompanied by exaggerated sensations of desire or repugnance — a *psychical hyperæsthesia* exists.

In all cases in which considerable increase of the sensible irritability takes place, the outbreak of subjective sensory excitement is also observed, that is, an excitement which appears without appreciable irritation of the peripheral sensory apparatus.

Accordingly there occur with great frequency in the hysterical, not only *pains* and *neuralgias*, but also *true hallucinations*.

Hyperæsthesia in the region of the organ of sight is sometimes so considerable that the patients are obliged to avoid every bright light, and as far as possible to betake themselves to darkened rooms. A peculiar sensitiveness to particular colors, especially to red, is observed in some hysterical people, who consider other colors pleasant. *Subjective light-phenomena* of a simple kind, flashes, sparks, and so on, appear occasionally before the attacks. More complex appearances, heads, figures, landscapes, etc., occur, as in the case of nervous persons, generally before going to sleep, but sometimes also during the day, as so-called phantasms, whose subjective nature is recognized without trouble. True *visionary hallucinations* appear particularly in attacks of ecstasy. These are more distinct and are often not recognized, even after the cessation of the attacks, as a subjective phenomenon. They may occur at the commencement of convulsive attacks, and occasionally also on the eve of all paroxysms.

When patients, in consequence of their abhorrence of light, remain for a lengthened period in dark rooms, there arises, from their dwelling in a feeble light, the capability of detecting variations in the latter. From this one must not, of course, conclude that an increase in the acuteness of vision has taken place in consequence of the disease, although such is undoubtedly the case in some instances. On the other hand, it is a matter of knowing or unconscious deception when hysterical people are said to have read with their eyelids quite closed.

Many of the hysterical are extremely sensitive to *excitations of the sense of hearing*. They are annoyed by any loud note, and often by the slightest sounds. In this condition they are frequently aware of events unobserved by the healthy, and, as with the sense of sight, a greater acuteness of the hearing may be artificially induced by the strictest avoidance of all possible sounds. But here also there occur cases of actual exaggeration of this faculty in consequence of the disease. As subjective sensations frequently met with, are to be mentioned ringing, blowing, roaring, and so on, which especially precede the spasmodic attacks. Phenomena analogous to the visionary phantasmata are rarer. But, again, true hallucinations of hearing, like those of sight, occur as well in ecstatic conditions as in other more complicated hysterical disturbances. They frequently constitute the transition which leads to definite mental aberrations.

The *senses of smell and of taste* are with especial frequency the seat of well-marked idiosyncrasies, whereby the capacity at the same time increases of detecting substances repugnant to the taste or smell, even when these exist in the smallest possible quantity. With this aversion to certain materials, which convey to others a pleasant scent or taste, the patients as a rule unite a peculiar preference for some tastes and smells, which are either a matter of indifference to the healthy, or to which they have an actual dislike. Thus it happens that the hysterical eagerly devour chalk, coal, sealing-wax, and other such things, and have a decided inclination for repellant odors, such as assafœtida, and the like. Cases, too, of actual increase in the acuteness of the sense of smell are known. Thus Amann relates the case of a lady who could detect the odor of cherries in a room, and who could distinguish persons by smell. Hallucinations of taste and smell are not rare in the simpler forms of hysteria. In more complicated cases they greatly encourage the development of erroneous ideas.

As in all these sensory regions, so also in the case of the *senses of locality* (Ortsinn), *touch* and *temperature*, a positive increase of sensibility is a comparatively rare phenomenon. In some well-attested cases, however, a refinement of the sense of touch seems to have been present, by means of which the patients could recognize persons and objects by feeling them, with an accuracy not met with in the healthy. In some cases too, as Eulenburg has correctly pointed out, the perception of a pulsatory movement at different parts of the body proves a condition of increased sensibility at these points—that is, when this movement itself is not exaggerated. Many of the cases, described in the literature of the subject as those of so-called refinement of the sense of touch, evidently rest upon a fallacy.

Yet in almost no hysterical person is there absent the symptom of *exalted sensibility to pain in some portion of the skin or more deeply seated parts, with and without spontaneous* *pains in the same.* The position and distribution of *cutaneous hyperæsthesia* varies extremely in individual cases. Sometimes it is distributed over the entire surface of the body, any movement is uncomfortable to the patient, and in some cases it induces general reflex convulsions. In other cases the skin of particular extremities only, of one half of the body, or of different portions of the trunk, is affected. Sometimes in the midst of hyperæsthetic regions there are irregularly circumscribed anæsthetic portions, or parts normally sensitive. The greatest increase of sensibility frequently exhibits itself on distinctly defined portions of the skin, especially on the head and back. On merely touching such places, the patients feel as if they were hurt, and were pierced with needles, or burnt with glowing iron.

Objectively there is as a rule nothing peculiar to be remarked in such portions of the skin, either as regards vascularity or in any other respect.

The spontaneous pains in the skin, which occur with and without hyperæsthesia, frequently assume the character of *neuralgia*, of which all known forms may arise as a result of hysteria.

Of more deeply seated parts, the muscles and fasciæ are very frequently the seat of hyperæsthesias and pains (myosalgiæ, myodyniæ); moreover, such affections do not rarely occur in the periosteum. In the latter case, if hyperæsthesia of the skin is not at the same time present, patients frequently only become aware of the condition in consequence of accidental pressure (knocks) on movement, or from examination specially directed to the part.

Painful affections of the internal organs are combined in many ways with those of the outer coverings. They will be considered together in the description about to be given of abnormal sensations in particular regions of the body, as on account of their immediate connection with spasmodic phenomena they must not be confounded with these.

Headache, in one form or another, appearing periodically, is absent in but few of the hysterical. After the attack many complain of a vague, confused feeling in the whole head, and with this there is frequently coupled a smart hyperæsthesia of the scalp, in consequence of which combing or gently pulling upon the hair becomes unendurable. The muscles which are attached to the galea aponeurotica, as well as this structure itself, may also be the seat of severe pain. Such also affects the temporal and cervical muscles. Moreover, in the hysterical, neuralgias of the different branches of the fifth and of the occipital nerves occur, and yet more frequently than these, hemicrania, which is very apt to appear at the menstrual period. An affection evidently very closely related to hemicrania is that which has been already described by ancient physicians as *Clavus hystericus*. Instead of the pain of hemicrania, difficult to localize and spread over the entire half of the head, clavus is characterized by a sharply defined, boring, gnawing, and burning pain, which, as a rule, has its seat in quite a limited area at the top of the head, somewhat to the side of the sagittal suture. Like hemicrania, this pain, otherwise not of frequent occurrence, makes its appearance very commonly at the menstrual period, and also as a result of trivial psychical excitations; it is accompanied by general discomfort, a feeling of faintness, sickness, and vomiting, and lasts, as a rule, for a few days, and in rare cases for several weeks.

Pains and abnormal sensations occur *in the region of the throat*, both in the skin and in more deeply seated parts. The most important of the latter, which have been grouped under the name of globus hystericus, must be mentioned along with the spasmodic affections. Hyperæsthesia of the mucous membrane of the larynx is sometimes so powerfully developed, that any current of air somewhat colder than usual, or slight admixture of dust in the air inhaled, produces severe pain and attacks of coughing. Here and there on the mucous membrane of the mouth and tongue, there likewise occur great sensitiveness and pain, without local changes being a demonstrable cause.

Painful affections in the region of the breast. Sometimes hyperæsthesia of the skin over the breast reaches such a pitch, that any touch becomes unendurable by the patient, and even moving the arms causes pain. Besides the hyperæsthesia, mastodynia, *neuralgia of the mammary gland*, is observed in some cases, and this, like the former, undergoes exacerbations, especially at the menstrual epoch. Much more frequently, however, than these comparatively rare affections, do *intercostal neuralgias* occur, and yet more often one finds some portion of the intercostal muscles, or particular ribs painful, without the presence of the typical symptoms of neuralgia.

At different parts of the *sternum*, too, and particularly in the neighborhood of the ensiform process, great sensitiveness to pressure is frequently met with. *Still oftener there occur in this region spontaneous pains of a heavy, dull character, which are usually coupled with a feeling of oppression and anxiety.* It is probable that these pains, which are frequently not to be localized with absolute precision, are caused in many cases by an *excited condition of the nerves of the heart.* Especially may this be assumed with tolerable certainty if the heart's action is at the same time exaggerated, as is usually found to be the case in the severer attacks of precordial anguish. In some rare cases of hysteria all the symptoms of *angina pectoris* are observed. Moreover, hyperæsthesia of different parts of the thorax usually induces more or less impediment to respiration, and it is evident that in this way also, and without any original implication of the nerves of the heart, a feeling of oppression may be caused. Besides, it so happens that a sensation of palpitation of the heart may subjectively be present in a very marked degree, without its being possible to determine the slightest sign of it objectively. In such cases it is possibly merely a question of hyperæsthesia of that portion of the chest wall against which the heart's apex strikes.

Painful affections of the abdomen. Universal and generally distributed pains over the entire belly appear most frequently in connection with the general tympanites to be described later, and as a natural consequence of it. Therewith one often finds a high degree of sensitiveness to the slightest touch, so that it is impossible for the patient to bear the pressure of garments or bed-clothes. Defective observation might in such cases lead to the erroneous diagnosis of a peritonitis, to which the absurd name of *hysterical peritonitis* has been given. In other cases hyperæsthesia exists without coming to the surface, and often indeed is demonstrably situated deeper than the skin. It is difficult to determine in such cases, where patients are extremely sensitive to any pressure upon the abdominal wall, and especially to the concussion produced by coughing and sneezing, whether we have to deal with hyperæsthesia of the peritoneum, as Valentiner believes, or with such an affection of the abdominal muscles. In circumscribed pains, too, in the abdominal region, one is dubious how much to attribute to the muscles, and how much to the viscera situated beneath them.

Simple *cardialgias* are of frequent occurrence, which, taken in connection with the persistent vomiting likewise often present, may lead to the erroneous diagnosis of round ulcer of the stomach. As the latter is also occasionally met with in the hysterical, caution in forming a diagnosis is always imperative. Some hysterical patients continually have *the feeling of a foreign body in the stomach*, which occasionally changes its position, and causes much anxiety. The *exaggerated feeling of hunger*, which recurs within a short time after meals, and frequently causes patients to take altogether incredible quantities of nourishment, appears to indicate an abnormally excited condition of the sensory nerves of the stomach. On the other hand, pains in the insertions of the straight muscles of the abdomen and of the diaphragm, rather than pain in the stomach itself, appear more frequently to be at the foundation of *the feeling of pressure and tightness in the epigastrium* met with betimes in most hysterical patients.

In the *hypochondria*, likewise, persistent pains and hyperæsthesias, which especially affect the muscles, are frequent phenomena. Many of the so-called inflammations of the liver and spleen, which the hysterical maintain they have so frequently suffered from, are merely to be referred to such conditions. Moreover, pains changeable in position and character occur in *the region of the small and large intestines*; these likewise are sometimes to be attributed to the external coverings, but at others must be regarded as true enteralgiæ. Especially interesting are the pains occurring in the hypogastric regions, which, in some cases, one can refer to affections of *the ovaries*. Even in the older writings, we find several such cases related. By the researches of Schuetzenberger, attention has afresh been directed to these symptoms, and Charcot[1] has, quite recently, minutely described them. In one case, Schuetzenberger[2] observed the outbreak of hysterical fits at the close of an attack of inflammation of the ovary, and could by pressure upon the painful ovary artificially produce these. In other cases, and in the complete absence of swelling and other inflammatory symptoms, he found the ovarian region very tender, and by pressure upon it, he likewise produced first pain and a feeling of tightness in the epigastrium, then the same sensations in the throat, whereon, by continued pressure, loss of consciousness and general convulsions followed. Charcot saw these phenomena supervene in like manner, and could further determine that, before the advent of unconsciousness, there occurred ringing in the ears and darkening of the field of vision; and that, too, at first upon the side corresponding to the painful ovary. The other phenomena also present in such cases (paralyses, contractions, anæsthesiæ, and so on) are said to have preponderated on the corresponding half of the body, and if the affection of the ovary changed its position, the other symptoms are likewise said to have crossed. On the whole, Charcot found the left more frequently affected than the right ovary. In accordance with somewhat numerous researches, recently instituted, I must contest the opinion that this phenomenon (which has been designated ovaralgia or ovaria) is one of frequent occurrence in the hysterical; when it is present it must be placed in the same category as the hyperæsthesiæ of other parts. Inflammatory conditions and demonstrable changes in the ovaries generally, as Schuetzenberger has already shown, are only exceptionally the cause, and the phenomenon, that by pressure upon the tender spot, an attack ushered in by an aura can be produced, is also observed in other hyperæsthetic parts in quite the same way. Schuetzenberger himself mentions some cases in which, independently of the ovary, the attacks could likewise be produced by pressure upon the epigastrium and upon the back. I have myself seen it induced several times by pressure upon tender vertebræ; and yet more frequently, on the occasion of an internal examination of the genitals, at the moment when the finger is passed through the vaginal orifice.

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[1] l. c., p. 283. A few other writings are referred to in the same place.

[2] l. c., pp. 748, 768, 829.

The *external genitals* are, in the hysterical, not rarely the seat of spontaneous pains and hyperæsthetic conditions. Especially at the menstrual period do some feel a very annoying irritation and burning in the labia and at the vaginal orifice, usually coupled with venereal desire. Sometimes these sensations, which, as a rule, are the cause of onanism, continue independently of menstruation.

Pain and hyperæsthesia in the *bladder and urethra* occur most frequently in such hysterical patients as suffer from actual diseases of the genital organs, and also as a result of the spasms which take place in these parts; sometimes, however, they appear as purely nervous phenomena, without any demonstrable cause.

Hysteralgia, as a purely nervous functional disorder, appears to be of extremely rare occurrence, while, on the other hand, pain and sensitiveness of the uterus, in connection with textural changes, are frequently met with in the hysterical. In particular instances one observes, what used to be considered the rule in hysteria, a direct dependence of the attacks upon uterine conditions; thus, attacks are occasionally perpetuated by changes in its posture, which disappear on improving the latter by means of pessaries, to reappear when these are removed; or severe pain and general convulsive attacks arise when diseased portions of the uterine mucous membrane are touched by the sound or therapeutic agents.

So-called *coccygodynia*—pain in the region of the coccyx, with great sensitiveness of that bone to the touch or to the contraction of the muscles in its neighborhood, seems only exceptionally to occur as a purely hysterical symptom, and must then be regarded as analogous to the hysterical vertebral pain.

Painful affections of the back are met with in most hysterical patients. The hyperæsthesia of the skin is sometimes quite confined to small portions over or between the scapulæ. The most frequent symptom, however, is *pain, spontaneous and produced by pressure, in the vertebræ and their surroundings.* The sensitiveness is sometimes limited to the spinous processes, and at others is more laterally distributed in the muscles of the vertebral column. If the skin itself does not participate in the hyperæsthesia, the patients frequently first remark the symptom when it is specially sought for. In other cases it appears as a result of considerable exertion—after prolonged walking or standing; finally, in yet other cases spontaneous back-ache is complained of. In these cases it is apparently a question partly of hyperæsthesia of the vertebral periosteum, partly of exaggerated muscular sensibility, and partly of true neuralgiæ. The latter are especially related to intercostal neuralgiæ, and the painful points to the side of the vertebral column are often none other than the points painful on pressure peculiar to such. One usually sees the symptom of pain in the back make its appearance alternately at different portions of the vertebral column; sometimes it is simultaneously present in vertebræ widely apart, and again it is fixed for a lengthened period in particular vertebræ. In the latter case, to the great injury of the patient, it has frequently been considered symptomatic of an affection of the vertebræ, or even of disease of the spinal cord. Especially, however, has it been, and still frequently is, looked upon as the sign pathognomonic of an irritative condition of the spinal cord —the so-called *spinal irritation*. There exists, however, no clear reason why in this respect back-ache should have any greater significance than neuralgiæ, myodyniæ, and hyperæsthesiæ arising in other portions of the body, which may all equally be considered phenomena proceeding excentrically from the spinal cord. The pain in the back also as little proves the old idea of the existence of a condition of hyperæmia of the spinal cord, as it does the more recent one of an anæmic condition of the same.

Hyperæsthesia of the skin and muscular pains, as already mentioned, are very frequently met with in *the extremities*. An excited condition of the sensory nerves of the muscles is, at any rate, at the bottom of a feeling which has been described as "uneasiness in the limbs." This is the same sensation as many otherwise healthy women experience at the menstrual period, and which is wont to appear in most people after great muscular exertion, and particularly after riding. It is possible, as Valentiner believes, that partial muscular contractions accompany this, but the peculiar sensation may also exist without these.

Finally, of especial interest are the *pains and hyperæsthesiæ occurring in articular regions*, which are sometimes mistaken for severe joint diseases. This affection, first described by Brodie, has had the peculiar fate of having been considered by some authors of frequent and by others of rare occurrence. Its existence has been altogether denied by some, while others have expressed the opinion that it may perhaps be a disease only met with in England. According to Brodie's description, the hip and knee-joints are most frequently affected, but it occurs in others, and especially in the wrist and finger-joints. Pressure upon the articular region produces severe pain; but this has its seat rather in the soft parts surrounding the joints, than in the articular surfaces, whose forcible apposition does not, as in true joint-disease, produce a violent shock. After a prolonged existence of the affection, which may continue for years, a slight swelling of the articular region may take place, which, however, is likewise distinctly situated in the surrounding soft parts. It does not produce actual deformity and muscular atrophy; but from the uninterrupted stretching of the muscle in the neighborhood an alteration in the shape of the joint may easily be simulated. The entire phenomena are sometimes liable to a rapid change. After continuing for years, they may suddenly disappear, in consequence of some moral influence or other. The fact that there are pure articular neuroses, and that not only in the hysterical, will scarcely be doubted at the present day. On the other hand, these are not with us, and as it appears also in France, among the frequent phenomena of hysteria. It may be questioned whether they can easily be mistaken for chronic articular inflammations; yet this mistake must have been very frequently made in Brodie's time, as he does not scruple to state, that "of women of the higher classes of society in whom joint-diseases are most commonly diagnosed, at least four-fifths suffer from hysteria and nothing else."

The same thing has occurred here as in the perfectly analogous case of affections of the vertebræ; pain and sensitiveness have been regarded as quite sufficient evidences of inflammation;

then the entire antiphlogistic armamentarium has, as a rule, been forthwith dragged to the rescue, and the patients condemned to abstention from movement, and thereby the disease has been aggravated so as to become persistent. Moreover, as Valentiner very properly points out, another error in diagnosis may be equally easily made, as that may be considered hysterical articular hyperæsthesia which is merely rheumatism in an hysterical patient.

Anæsthesia.

Diminution or loss of sensibility, temporary or persistent, may arise in the hysterical in the region of all the senses. Anæsthesia of the *sense of touch* is the most frequent; but Gendrin, who first more precisely described this affection, goes much too far when he maintains, that in every case of hysteria, from beginning to end of the disease, general or partial loss of sensibility is present. As little can I confirm the assertions of Henrot and Szokalsky, that after hysterical attacks, without exception, anæsthesia in one part of the skin or another may be demonstrated; when such is the case, the examination may have been limited to that period of the attack in which semi-consciousness and apathy prevail.

It is, however, true that anæsthesia of the skin and of the deeper parts appears most frequently after hysterical attacks, and that, as a rule, if it has diminished in the interval between attacks, it is again aggravated by a fresh fit. Szokalsky states that it occurs in a more pronounced form and more universally, after more severe than after milder attacks. Extensive and persistent anæsthesia may, however, disappear after a fresh attack; further, in place of the part at first affected, others may be attacked, or the part formerly anæsthetic may become hyperæsthetic.

In the majority of cases in which anæsthesia occurs, sensibility to pain alone is diminished or removed, while the sensation of pressure and heat is normal; cases in which all sensation is absent are of less frequent occurrence. In these, moreover, manifold differences occur, as sometimes the appreciation by the skin of distance (Raumsinn) and of pressure (Drucksinn) suffers loss, but variations of temperature are correctly felt, while in other cases the perception of this, too, is completely lost.

Anæsthesia extending over the whole surface of the body is most rarely observed; that limited to one half of the body, hemianæsthesia, occurs rather more frequently, the left side, according to the statements of Briquet and of Charcot, being attacked by preference. Anæsthesia is most frequently met with in some of the limbs or in a portion of them. Especially often is it situated on the dorsal surface of the hands and feet, as well as in the region of the outer ankles; sometimes it appears isolated at different parts of the skin. With the more widespread form of anæsthesia of the skin, a similar condition of the mucous membranes is usually coupled, in which the normal reflex actions can then no longer be excited. In cases of unilateral anæsthesia, one finds the mucous membrane only of the affected half of the body insensible. Sometimes the vessels of anæsthetic portions of the skin are persistently contracted. Such parts usually appear pale and cold, and it is difficult to succeed in causing a flow of blood by pricking with needles.

It has been assumed aright, that in many known cases in which hysterical persons have intentionally mutilated their bodies in the most revolting manner, pushed a number of needles under the skin, swallowed pointed bodies, or inflicted deep burns, *analgesic states* have been present, in consequence of which those mutilations have been painless.[1] But it is also possible that the peculiar mental condition of hysterical patients may be the cause of their ignoring pain actually felt, and appearing not to feel it. The desire to create a sensation and to render themselves interesting from their condition may induce such a morbid increase of the power of will that the most excruciating agonies may be borne with a stoical indifference. Such patients are as a rule very willing to serve as subjects for physiological investigations, and under such circumstances allow themselves to be tortured with the most powerful skin-irritants and the strongest electric currents, if they only believe that thereby they become interesting or acceptable to the investigator. To the question, "Do you begin to feel pain?" they answer, "Not in the least; I can easily bear it."

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[1] In one such case which was recently under my care this could be directly proved. An hysterical woman, who had formerly suffered much from general convulsions, and had for years complained of pains of all kinds in consequence of hysteria, fell without known cause into a state of violent excitement, in which for the first time during the course of her disease many extremely painful hallucinations of hearing and sight appeared for a few days. In this condition she managed one day, in the absence of her nurse, to open the door of the oven, and to extract with both hands glowing embers, which she pushed firmly into her sexual parts. A deep burn on the inner surface of the hands, as well as on both labia, and the inner surface of the thigh, was the result. The patient, whose hallucinations ceased from that moment, afterwards maintained that she had wished in this peculiar manner to take her life; and that *she had no pain while being burned either in the hands or genitals*, although she had distinctly felt the heat of the embers. During the slowly progressing cicatrization, too, no pain occurred in these parts, usually so sensitive.

With the loss of cutaneous sensibility, a similar loss of sensibility in the muscles,[1] *as well as in the bones and joints, may be united.* In this case patients lose the power of appreciating with closed eyes passive movements imparted to their limbs. Thereby the power of spontaneous muscular contraction may remain normal or be altered in varying degree. Sometimes this kind of anæsthesia is coupled with paralysis, at other times with catalepsy. In the case of an hysterical patient here who suffers from cataleptic fits, together with the flexibilitas cerea, which is as a rule limited to one arm, there is complete loss of sensation in this arm, so that the patient neither feels the most painful impressions, nor is she with closed eyes aware of what is done with her arms. With the subsidence of the cataleptic state the anæsthesia is also wont in this case to disappear. In anæsthesia of the ocular muscles Szokalsky saw the regulative (mimischen) movements of the eyes suffer in a high degree.

Anæsthesia of the mucous membranes, usually appearing in connection with the general cutaneous anæsthesia, sometimes also occurs alone, and is then generally coupled with diminution or loss of reflex excitability. Thus foreign bodies may come in contact with the anæsthetic conjunctiva without producing reflex spasm, lachrymation or reddening. In anæsthesia of the nasal mucous membrane, it is not possible, by tickling with a feather or brush, or by means of powerfully irritating substances, to give rise to sneezing, and the pharyngeal mucous membrane may be equally titillated without the occurrence of vomiting. In such cases the senses of smell and of taste may likewise be abolished, or remain unaffected. Further, besides an hyperæsthesia of the respiratory tract, an anæsthesia of the same has been described, as a result of which irritating vapors (of ammonia, sulphur, etc.) may be introduced into the larnyx without causing cough. It is scarcely possible to state whether anæsthesia of the bowels occurs, as these parts are normally endowed with a low degree of sensibility. On the other hand, one frequently finds them hyperæsthetic in cases of extensive anæsthesia of the skin and muscles. The abdominal wall may then be insensible, while deeper pressure in particular regions calls forth severe pain.

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[1] See Vol. XI. of this Cyclopædia, p. 233.

The collection of large quantities of fæces in the rectum, which sometimes occurs in the hysterical, seems in some cases to be induced by anæsthesia of the mucous membrane in question.

The mucous membrane of the genital organs and of the urinary passages is likewise found to be insensible in some hysterical patients. In the latter case, the filling of the bladder is not observed, and this may go on to great distention of the same, and secondarily to dribbling; in such a case the passage of the catheter through the urethra is not felt. Complete anæsthesia of the vaginal mucous membrane, with consequent absence of all sexual desire in copulation, Scanzoni could only determine in four of his numerous patients, in whom, besides, feeling was occasionally blunted only on the dorsal aspects of the hands and feet. This phenomenon, according to Amann, occurs much more frequently.

I have already adverted to a case of diminished sensibility to pain in the external genitals. On the other hand, as a case of Scanzoni's proves, a universally distributed anæsthesia of the skin may occur without anæsthesia of the genitals.

Finally, anæsthesia may arise in the other sensory regions with and without concomitant cutaneous anæsthesia. With the one-sided occurrence of the latter there sometimes exists loss of taste in the corresponding half of the tongue, and of smell in the corresponding nostril, as well as unilateral amblyopia and deafness.

Of visual disturbances *amblyopia*, as well as complete *amaurosis*, is observed. Both as a rule appear after severe hysterical attacks, but may also develop themselves in the absence of these. According to the statements of Charcot and Galezowsky, the condition occurs unilaterally, pretty often, and indeed generally in conjunction with hemianæsthesia of the same side, although the patients themselves as a rule are not aware of this. Galezowsky found a state of "*dyschromatopsia*," loss of the power of distinguishing color, to be the lowest degree of amblyopia; then he noticed a partial limitation of the field of vision, partly in its peripheral portions, and partly so that one-half was dark (and, in the left eye, the outer half of the corresponding retina), so that in the one eye there existed a condition of *hemiopia*, while in the other the sight was normal. Amblyopia and amaurosis in both eyes are rare events. I myself have found the former distinctly pronounced only in one eye, in which it repeatedly arose after severe hysterical attacks, on each occasion again to disappear after a few days. In ninety-three cases of anæsthesia in different sensory regions, Briquet found amblyopia six times.

Ophthalmoscopic examination generally discloses no changes, and this was so in the cases observed by me. In a patient in Charcot's ward, in whom amblyopia had existed for a long time without ophthalmoscopic changes, Galezowsky, however, saw an infiltration and capillary reddening of the disk, with fusiform dilatation of the arteries, finally make its appearance, and in another, as Svynos relates, the rapid invasion of atrophy of the optic nerve could be determined. It is questionable indeed whether, in the latter case, one could still speak of hysteria.[1]

Hysterical *deafness* is likewise occasionally observed in severe cases of hysteria, and that too in one ear as well as in both, and in the absence of any demonstrable disease. It also generally continues after severe attacks, and combines with other anæsthesiæ. Like hysterical blindness, it may suddenly disappear, and be supplanted by other hysterical symptoms.

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[1] Consult in particular upon this condition, besides works in general on hysteria, in which hysterical blindness is fully considered, *Charcot*, Leçons. etc.; *Mesnet*, Thèse sur les paralysies hysteriques 1852; and *Svynos*, Thèse sur les amblyopies et les amauroses hystériques 1873.

Motor Disturbances.

1. Convulsions.

The convulsions occurring in hysteria are to be regarded partly as reflex, and in part as arising without the recognizable co-operation of a centripetal stimulus.

To the former one may also in a wider sense allot those caused by mental excitation, which in their typical gradation (Abstufung) and spread show a mere exaggeration of the involuntary movements called forth by the emotions in health. We shall begin with a description of the forms of spasm met with in particular sections of the muscular system, in order thus to learn their manifold combinations in the so-called fits. An abrupt distinction of reflex spasms, from those due to a direct excitation of motor parts, can neither be carried out in this exposition, nor is it always possible in practice.

Spasms in the Digestive Canal.—The most frequent form is that which occasions the so-called globus hystericus. This symptom is only absent in a few of the hysterical, but in individual cases is very variable in its character and in the frequency of its recurrence. Patients usually have the sensation as if a foreign body, generally of the shape of a ball, passed upward from the stomach or sôme part of the digestive tube, and remained impacted in the region of the throat. The ball frequently arises in this part, without changing its position further. After continuing for a longer or shorter period, the feeling gradually becomes less marked, or suddenly vanishes. The ball seems to flatten. Sometimes, instead of this sensation, only that of a tightening in the throat exists, similar to that which so easily develops itself in the healthy from emotions of anxiety or fear. Some patients do not feel the movement of a ball, but of some other differently shaped body, such as a kernel, a bean, and so on, or of an animal creeping about in the throat—of a worm, as one of my patients constantly affirms. Not rarely the sensation of something present is so distinct that patients think they can pull it out of the throat with their fingers. The entire phenomenon is apparently caused by peristaltic contractions of the œsophagus passing upward (less frequently the movement is observed to be reversed—the ball descends), and partly by a circular tightening of the pharyngeal muscles.[1] It sometimes arises spontaneously, especially as the forerunner of a general convulsive attack; it is, further, the most usual accompaniment of psychical excitement in the hysterical, and finally, often develops itself in an evidently reflex manner. The latter regularly occurs in some patients while eating; occasionally particular kinds of food produce the bolus, and now solid, and at other times fluid food is most easily borne. In some cases the symptom is so strongly pronounced that it appears with every attempt to swallow, and compels the sufferer to take almost no nourishment, so that one has to employ the œsophageal tube to feed the patient. In others the globus is induced by the mere exertion of speaking or singing. An hysterical lady whom I know has had to abandon her singing exercises entirely, as, with every attempt she makes, the ball in her throat is felt to rise, and does not as a rule subside for some hours. In such cases it is sometimes sufficient merely to think of the causal irritation to call forth the spasm.

With the spasmodic symptoms in the pharynx a similar condition of the tongue is occasionally combined. With each attempt at movement, the latter is violently and irregularly contracted, being drawn sometimes to the side, at others upward, and again pushed backward, so that articulation as well as swallowing may be materially impeded.

The globus occurs not only in the throat, but also in the abdomen, as the sensation of a body rising from the region of the symphysis toward the stomach. Some patients aver that they distinctly feel the womb pass upward to the stomach, and then arrive at the throat, a declaration which has constituted the basis of the oldest theory of hysteria. Probably this has, as a rule, been due to abnormal sensations in the genitals, causing reflex contractions in the stomach, œsophagus, and pharynx. Sometimes, also, powerful peristaltic movements occur in particular parts of the bowels, which are felt through the abdominal wall, and still further convince the patient of the presence of a movable body. Spasmodic strictures in different portions of the small and large intestine may, moreover, arise alone and cause local or general swellings. When such local swellings continue for a length of time the appearance of a tumor of the bowel, or other abdominal organ, may be given rise to, especially if fæcal masses accumulate at the constricted point, and these do not reveal their true nature by a tympanitic note. Spasmodic phenomena occur in the stomach as one of the symptoms of cardialgia. They are seen most evidently in so-called hysterical vomiting. Patients who suffer thus, usually vomit after each meal, the food reappearing very soon, almost quite undigested. Less frequently, too, mucous and bilious matters are vomited while fasting. The appetite often suffers only slightly in consequence; the patients try ever anew to take fresh nourishment, notwithstanding the hopelessness of the attempt, and have generally an especial desire for fluids. In other cases, again, the appetite fails completely, and then, as the symptom usually persists for many months, well-marked conditions of debility arise. But, when the patient apparently stands on the brink of the grave, a sudden change almost always takes place; the symptom which has bid defiance to every remedy disappears of its own accord, and the patient rapidly recovers. Concerning the hysterical vomiting of blood, which is, as a rule, unconnected with this habitual and continuous vomiting, see further on, as also concerning the vomiting which accompanies an hysterical retention of urine. The form here described has evidently the greatest similarity to the vomiting, frequently so obstinate, which appears during the early months of pregnancy; like that, it arises in a pre-eminently reflex manner, soon after the introduction of substances into the stomach. The obstinate eructation also, which is often met with in hysterical persons, especially after the attacks, is mainly a reflex act, and is caused by a great development of gas in the stomach, but also occurs without palpable or visible distention of the gastric region.

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[1] At all events I cannot convince myself of the correctness of *Eulenburg's* view, that we have not here to do with motor, but merely with sensory phenomena.

Spasmodic movements in the organs of respiration.[1]—There sometimes occurs in the hysterical a temporary acceleration and exaggeration of breathing, without giving rise to a feeling of embarrassed respiration. Further, temporary spasmodic pauses in inspiration and expiration take place, especially in conditions of psychical excitement. Moreover, one sees true asthmatic attacks arise, with an acute sense of oppression and anxiety, and with distinct interference with expiration. Occasionally it is observed that these symptoms, due apparently to spasmodic contraction of the bronchial muscles, may be induced in a reflex manner by a diseased uterus, either in consequence of force employed in examination, or by irritation of the inflamed mucous membrane (asthma uterinum).

Singultus occasionally sets in as a very annoying symptom after hysterical attacks. In some cases this has been seen to continue with great severity for days. Much and irresistible *yawning* is likewise observed in attacks of greater or less duration. *Convulsive laughter* and *weeping* may usher in or follow the general attacks, but may also arise alone as independent fits. It may frequently be shown that some corresponding psychical impression originally formed the basis of these conditions. Not only is this, however, on the whole quite unimportant, but these paroxysms, extremely painful in their more pronounced manifestations, often enough arise without any psychical cause.

The vocal muscular apparatus may also be thrown into a state of spasmodic excitement. Loud screams frequently occur at the beginning of, and during the attack, and some patients thus imitate the cries of animals by miawing, barking, howling, and so forth. Such screaming fits are observed with especial frequency during the epidemic spread of hysteria. Moreover, in the intervals of the attack, sometimes while speaking, an inarticulate sound or involuntary exclamation annoys the patient. Of the frequently occurring spasms of the laryngeal muscles, the spasmodic closure of the glottis is of especial importance, as it often produces alarming suffocative symptoms, and in rare cases has even caused death. This form of spasm is also coupled with attacks elsewhere, especially with the tetanic form of the same; it can, however, also occur alone, and accompanied merely by the corresponding dyspnœic movements.

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[1] Consult the exhaustive description of these phenomena in Volume XI. *Erb*, Peripheral neuroses.

Spasms in the urinary and generative organs.—Spasmodic retention of urine occurs in many hysterical patients, particularly in connection with painful affections of the genitals, but also without demonstrable change in these. Increased inclination to micturate is often combined with this. On introducing the catheter into the bladder, a considerable obstacle has to be overcome in such cases. The great irritability of the vesical muscle is shown in some hysterical cases by the very frequent inclination to micturate without the existence of increased secretion, or impediment to evacuation. Spasm of the constrictor cunni, which possibly always arises in a reflex manner, and is the cause of so-called vaginismus, has already been mentioned in speaking of hyperæsthesia of the vaginal orifice.

Besides those already described, local spasms may take place in all possible sections of the muscular system of the head, trunk, and extremities, as a symptom of hysteria. These occur as clonic or tonic spasms, sometimes in fits, at other times persistently in the territory of one or several peripheral motor nerves. Their more minute description may here be dispensed with by referring to the chapter on this subject in volume XI. But there yet remains to be mentioned the generally distributed muscular uneasiness of frequent occurrence in the hysterical. Here we have to do partly with the spontaneous contractions, now here, now there, either affecting an entire muscle or some of its groups of fibres, and partly with combined movements, which appear either as direct or reflex, and which may also occur more universally in consequence of the slightest mental disturbance. The facial muscles especially are incessantly active in many of these patients, so that the physiognomy has a somewhat restless and unsettled character, which has been considered by some authors to be an infallible sign of hysteria. From the further progress of this general muscular uneasiness, there proceed either attacks of general clonic convulsions or the symptoms of chorea minor, which is not rarely observed as a particular incident in severe attacks of hysteria. The description of general convulsive attacks is given further on. A peculiar form of spasm likewise, permanent contraction, can only be removed after paralysis.

2. Paralyses.

Paralytic conditions of the muscular system of the bowels and stomach are, if not the cause, at any rate the usual consequence of the local and general tympanites which is so frequently met with in the hysterical. The latter phenomenon arises more frequently at the close of hysterical convulsive attacks, but also occurs independently of these. It frequently forms within a very short time, and by no means always during the period of digestion. It comes on very rapidly, especially after mental agitation, and at other times without a known cause. In some patients I have seen it appear for a lengthened period regularly every morning, and reach its climax about mid-day. Sometimes it attains such a degree, that the patients can be kept afloat in a bath by means of the balloon-like distention of their bellies. The appearance generally vanishes after having lasted for a short time, as by the powerful action of the abdominal walls, or the contraction of the resuscitated muscles of the stomach and bowels, a forcible evacuation of gas takes place from above downward. Occasionally, however, it persists for days. The patients are unable to effect a spontaneous evacuation. In such cases one can often cause the whole stomach to collapse within a short time, by forcible pressure upon the abdominal walls, or by powerful faradization of the same, when, with an audible sound, gases rush from mouth and anus. In other cases the introduction of the intestinal tube is necessary to give vent to the flatus.[1]

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[1] In the great hysterical epidemics of former centuries, tympanites was commonly observed at the close of paroxysms. The patients were relieved by bandaging the abdomen tightly, by striking it with the fists, or treading upon it with the feet, and thus the accumulated gases were evacuated.

The gases are, as a rule, almost quite odorless, whence it may arise that in cases in which they inaudibly escape from the anus, a disappearance of the tympanites without the escape of flatus is apparently observed. This is at all events much more probable than that a rapid absorption of a large quantity of gas by the intestinal wall should take place. Nothing certain is known concerning the source of this so frequently sudden development of gas or of its composition. The distention is undoubtedly sometimes caused by swallowing air, at which some are known to be very expert. The excessive distention may give rise secondarily to a paralysis of the walls of the stomach and bowels, which renders a spontaneous expulsion of flatus impossible. But in other cases it can only arise from the contents of the intestine. Whether it can also be generated by the blood circulating in the walls of the intestine is doubtful.

Paralytic or paretic states of the muscular system of the bowel are also often the apparent cause of the extremely obstinate constipation met with in the hysterical.

Paralysis of the muscles of the œsophagus and pharynx also occurs as a symptom of hysteria. Swallowing is thereby rendered difficult, or altogether impossible, while the œsophageal tube introduced glides without obstruction into the stomach. This condition, which torments the patient exceedingly, and by interfering with nutrition induces a high degree of exhaustion, it is frequently possible to cure almost at once, by using the tube, or by employing electricity or other stimuli. In other cases feeding must be undertaken with the tube for some time, and improvement takes place with the removal of the general debility.

Paralysis of the vocal cords is the most frequent of hysterical paralyses met with in the respiratory organs. This generally appears suddenly, sometimes after an attack, but more frequently without such, and is then chiefly induced by some psychical excitement. The patients either completely lose the power of speaking aloud or the voice seems husky, disappearing with any considerable effort. Sometimes, while engaged in lively conversation, a sudden loss of voice ensues. With this *hysterical aphonia*, difficulty in moving the tongue may be united, so that patients can no longer even whisper, but can only make themselves understood by signs.

Examination with the laryngeal mirror shows vocal cords quite normally colored, in simple hysterical aphonia, and in the vast majority of cases exhibits paralysis of the constrictor glottidis, sometimes on one side, at other times double. The musculi thyreo-arytænoidei interni are often paralyzed; this is, however, more rarely the case with the dilatator glottidis. Hysterical aphonia may disappear as rapidly as it supervened. Sometimes psychical excitement is the remedy, at others the most varied methods of local treatment:—external manipulation of the larynx, the introduction of the mirror, systematic lessons in intoning, electricity applied externally or directly to the affected muscles. There always, however, remains a tendency to after-effects, which is proved by the fact that after repeated recurrence, the paralysis does not disappear so readily as it did at first; some cases bid defiance to all treatment for years, until they are finally cured by some accident.

Paralyses of particular muscles of respiration occur, like other circumscribed hysterical paralyses, but they are rare. Temporary paralysis of the diaphragm, which one sometimes sees after epileptic, may possibly occur after hysterical fits, but I am ignorant of any such observations. *Paralysis of the bladder* is met with in the hysterical, both secondarily, in consequence of spastic retention of urine, and also as a primary symptom.

Paralysis in the extremities.—This sometimes appears in a hemiplegic and at others in a paraplegic form; now only one extremity is affected, and again there is crossed paralysis of an upper and the opposite lower extremity; in some cases total paralysis of all four extremities has been observed. Of the facial muscles, those acting upon the eyeball only very rarely participate in the paralysis. On the other hand, paralysis of one levator palpebræ superioris, or of both these muscles, is met with pretty often. Moreover, paralysis in the territory of the facial nerve and of the hypoglossal is extremely rare. In otherwise perfectly complete hysterical hemiplegia, drawing of the face towards the healthy side, so constant in apoplectic hemiplegia, and the pushing of the protruded tongue to the paralyzed side, can almost never be determined.

Besides paralysis affecting entire limbs, such a condition is met with in the extremities in the territory of particular motor nerve trunks; these are, however, rarer than the more general paralyses. The degree of paralysis may be very different, beginning with mere weakness and heaviness of the limb, to absolute loss of motion. The commencement is often sudden, as the paralysis is left fully developed after an hysterical attack; in other cases it comes on gradually without a preceding fit: patients complain of a feeling of weakness in one or other extremity; this shortly increases in intensity and extent, until finally a greater or less degree of paralysis is established. *The behavior of the paralyzed muscles to the electric current is normal;* the excitability both for the induced and for the constant current remains unaltered, even after a persistence of the paralysis for years; it is only on account of the atrophy induced by long disuse, which, however, never attains a high degree, that the response to electricity may at a later period be less energetic.

In the great majority of cases a certain degree of anæsthesia develops itself in the paralyzed parts; but not by any means so that the amount of each condition stands in any definite relation. Total anæsthesia may occur without paralysis, and, though rarely, the converse may also be the case. If anæsthesia is present, the muscular parts also, as a rule, participate, and in consequence of this, sensation on electric contraction of the muscle is absent. Duchenne has proposed this condition of diminished "electro-muscular sensibility" as an infallible sign of hysterical paralysis, by means of which one may distinguish this especially from apoplectic palsy. Against this it must be remarked that anæsthesia, at least to any considerable amount, may be absent in hysterical paralysis, and that where cutaneous sensibility exists, it is simply impossible to estimate exactly the degree of electro-muscular sensibility, and moreover, that in apoplectic paralyses anæsthesia is also sometimes met with, and that, too, with complete loss of muscular sensibility.

If this diagnostic sign is therefore only applicable to some cases, it is, as a rule, possible to recognize the character of the paralysis from other symptoms present. Hysterical paralysis does not appear unless other and distinct signs of hysteria are coexistent, such as spasmodic attacks, with and without reference to the paralysis present, hyperæsthesia in the most varied parts, and particularly the characteristic psychical symptoms. A paralysis, then, which appears in an extremely hysterical subject, must always suggest the possibility of its being a symptom of hysteria.

In doubtful instances the progress of the case furnishes information: hysterical paralysis is sometimes of very short duration, continuing for a few hours, days, or weeks, after an attack, and then completely disappearing, perhaps to return after subsequent fits. If these conditions have already frequently shown themselves, one will rarely be in doubt as to the diagnosis. In other cases the mode of extension of the paralysis is characteristic ; it appears crossed from the commencement, or it is at first pronounced on one half of the body, quickly disappears from hence, and suddenly shows itself on the other side, and possibly also makes several such changes of position. But there are other cases in which the paralysis continues for years unchanged in extent, and in these cases the diagnosis cannot with absolute certainty be made for a long time. It may especially be confounded with hemiplegia from disseminated (Herderkrankungen) brain disease, and with paraplegia, the result of disease of the spinal cord. The latter error often arises, particularly in those cases in which in paralyzed hysterical patients decided hyperæsthesia, due to disordered sensibility, is at the same time present in the vertebral region. The skin of the back of such patients, covered with cicatrices, is frequently an eloquent witness of the diagnosis formed, and which was followed by an active derivative treatment. It must nevertheless be acknowledged that the heroic remedies of burnings and caustics now and then cause an hysterical paralysis to disappear just as suddenly as any other remedy which is employed against the disease, or as some accidental, purely psychical influence. Finally, the most important criterion for making a diagnosis is the sudden disappearance, through the agency of a moral influence, of a paralysis which had continued for years, and bade defiance to all treatment. The diagnosis may, however, be attended with peculiar difficulty in those cases in which it has to be distinguished from multiple sclerosis of the brain and spinal cord—a disease in which likewise a frequent change of locality of the paralysis, a coming and going of it, is often observed, and whose attacks of an apoplectic character may be deceptively simulated by hysteria. The diagnosis of severe forms of hysteria from this disease is often first made after long-continued observation.

A condition of *permanent contraction* not rarely develops itself in the palsied extremities. In some cases this appears simultaneously with the paralysis. After a convulsive attack, one extremity or half of the body, or both upper or lower extremities remain in a state of tonic contraction, which cannot at all be overcome by the will of the patient, and passively only by great force.

In other cases paralysis continues for a length of time, and then gradually or suddenly contraction supervenes after a fresh attack. In the upper extremities spasmodic flexion of the forearm, hand, and fingers is almost always present. In the lower extremities, on the other hand, according to Charcot, who has of late redirected attention to this affection, spasmodic extension at the hip, knee, and ankle-joints is the rule, with which contraction of the femoral adductors may also be associated. To this rule there are, however, exceptions, as the following case, noted by me in Würzburg, proves:

In a cook, twenty-eight years of age, who had, some time before her reception into the Julius Hospital, suffered at her own home from a tedious abdominal affection (probably purulent oophoritis, with bursting of an abscess into the rectum), together with peculiar psychical symptoms, there developed itself powerful persistent tremor of the left leg, which hindered the patient in walking. A few weeks later general convulsive attacks made their appearance, in which the right leg was thrown into a state of tonic extension, and the left into tonic flexion, while violent clonic convulsions followed in the facial and lingual muscles. These very frequently repeated attacks, coupled with complete loss of consciousness, usually lasted for one or two minutes: they terminated in a soporose condition, which quickly passed off. After the attacks, the rigid extension of the right leg completely disappeared on each occasion: the flexion of the left, on the contrary, very soon became permanent, and in such a manner that the leg at the knee-joint was bent at a right angle, and could not be straightened by exerting the greatest force. But the semiflexion of the femur and the clubbed position (varus) of the foot could be easily adjusted. The contraction of the knee could only be overcome when most deeply under the influence of chloroform. After persisting for about a year, this as well as the anæsthesia present in the same leg and the hysterical fits disappeared, on the occasion of the patient being attacked by typhoid fever. During the following three months none of her numerous hysterical symptoms returned, of which, oft-repeated vomiting of blood, great salivation appearing in paroxysms, tympanites, and nervous deafness, may be mentioned. The patient then died of rapid tuberculosis of the peritoneum, the source of which was to be referred to chronic inflammation of the Fallopian tubes and the structures surrounding the uterus. Both ovaries were atrophied, and the tunica albuginea thickened. The stomach was free from ulcers and cicatrices; in the bowel numerous slaty spots were recognized as the remains of the former typhoid affection. The brain and spinal cord were microscopically healthy.

Contraction, like paralysis, may last for years, and like this may suddenly be cured by all possible moral influences. But there are also cases which resist every influence, and in which, after continuing for decades, atrophy of the affected muscles with diminished electric response finally sets in. Under the deepest influence of chloroform the contraction can then no longer be overcome.

It is feasible to suppose that in such cases the at first purely functional disorder may later go on to material changes in the nervous system, that is, that the supposed condition of excitement, of whose material basis we are ignorant, after continuing for a lengthened period, is accompanied by grosser changes at its place of origin. Charcot has related a case in which this may have occurred. In a female hysterical patient, who had suffered for ten years from contraction in all four extremities, which had at first disappeared several times, but later became permanent, sclerosis of both lateral columns was found on examination. Against this it may be urged that the sclerosis was in this case the original disease, merely by chance complicated with hysterical symptoms. At any rate, as Charcot himself says, it is impossible to state the exact period at which sclerosis develops itself in a case of hysterical contraction.

As in contractions and palsies, caused by anatomical changes in the brain and spinal cord, so in hysterical conditions of this kind tremor is frequently observed, which appears in the paralyzed extremities, especially on attempting movement. But this may also occur as an independent symptom of paralysis and contraction, and then appear in quite the same way as in paralysis agitans. An analogous shaking palsy in the head is also sometimes observed. Less marked degrees of tremor may, moreover, occur in the tongue, the facial muscles, and the hands, in most hysterical patients, as quite transitory phenomena, which may be called forth by the slightest psychical excitement.

Disturbances of the Circulation, and Disorders of Secretion and Excretion.

Changes in the power and frequency of the heart's action are often met with in the hysterical. The disturbances are, as a rule, transitory, less frequently permanent. In most patients it only requires trivial psychical or sensory excitants to produce powerful *palpitation*, which is felt not only subjectively by the patient, but may also be appreciated objectively, as the apex-beat seems increased, and strong synchronous pulsations are sometimes found in the whole cardiac region. The frequency of the heart's action is almost always at the same time increased.

The pulse in such cases is sometimes small and hard, at other times full and soft, corresponding to the varying state of contraction of the muscular coat of the vessels. The more or less complete paralysis of this coat often rapidly succeeds a temporarily excited condition. In place of the paleness existing at first, general reddening of the skin and profuse perspiration set in, and at all possible portions of the body, at which no pulse is at other times to be felt, vascular pulsation, appreciable subjectively and objectively, occurs. Such transitory exaggerations of the heart's action are also found in the hysterical, without a demonstrable psychical or sensible cause; they are also not very rare symptoms in the course of hysterical convulsions. In the latter, moreover, the heart's action is not by any means always increased; much more frequently, in contradistinction to the spasmodic movements in all other muscular parts, its normal activity is diminished. A feeling of fullness and oppression in the chest, with anxiety, is a regular accompaniment of palpitation. Its influence upon consciousness (Gehirnthätigkeit) is very variable, and in accordance with the conduct of the vessels of the brain at the same time. A feeling of confusion and oppression in the head, as well as giddiness, is only rarely absent in severer attacks of palpitation.

Considerable *weakening of the heart's action*, and especially such as supervenes suddenly, is also met with in the hysterical. This may usually be determined in the fainting fits which so easily arise in some of these patients, and together with the accompanying anæmia of the brain apparently causes these attacks. The occurrence of the latter is favored by a simultaneous and general diminution of vascular tonus, which, in the presence of a weakened heart's action, has as a necessary consequence an accumulation of blood in the more deeply situated parts of the body. The diminished heart's action is likewise very remarkable in attacks of catalepsy, but seems to reach its highest degree in those rare comatose states which last for days, and which have been designated hysterical trance.

In the ordinary state, apart from all fits, the action of the heart and of the pulse is in many hysterical persons perfectly normal, as regards both frequency and force. An habitual weakness of these occurs in those by no means rare cases in which chlorosis and anæmia exist as causes or concomitant symptoms of hysteria. The real blame of the occasional disorders of cardiac and vascular action mentioned is often apparently to be attributed to these conditions.

Independently of the state of the heart's innervation, *variations in the state of contraction of the muscular coat of the vessels* occur in a great many hysterical patients, producing remarkable appearances, particularly in the vessels of the skin. As an habitual condition, one finds with especial frequency pale and cold extremities, while the face is much reddened, and the patients complain of an overwhelming sense of heat in the head. At remarkable variance with this, the mucous membranes are sometimes bloodless, both the conjunctiva and lips being very pale. Even when the color of the face is usually normal, one generally notices in the hysterical a great tendency to become pale and to blush. With the reddening of the skin, profuse perspiration of the face then, as a rule, takes place. The same appearance is also met with at other portions of the cutaneous surface. Thus, the hands especially, which when at rest are dry and cool, often become warm and moist on the slightest attempt at manual labor, such as writing and the like. This is also the case in the lower extremities. In other cases perspiration is increased over the whole body; cases of unilateral sweating are also occasionally seen in the hysterical. Local hyperæmia of the skin does not rarely accompany the neuralgic affections which occur in hysterical persons. Herpes, too, is sometimes observed along with it. In the extremities, in which the joint affections previously mentioned arise, Brodie frequently observed the typical variations of vascularity and temperature take place, in such a manner that icy coldness and paleness of these existed for some hours daily, then elevations of temperature, with reddening and sweating appeared, which continued for about the same period, and then for a time gave place to a normal condition.

Of especial interest are *the bleedings from the skin and various internal organs* met with in hysterical persons, in consequence of local congestion. To estimate this aright, *the state of menstruation* must next be considered. This is, in many hysterical women, perfectly normal; yet in nearly a half of such patients disorders of this function of one sort or another occur: sometimes bleeding, profuse and frequently repeated; at other times abnormally little, and only occurring at long intervals. Sometimes these are undoubtedly the result of anatomical changes in the genital organs; at other times, whether appearing with or without such changes, they are, in the manner previously stated, the cause of hysteria. Amenorrhœa may further be the mere result of the anæmic condition causing the hysteria; it may, however, in the absence of anæmia, also arise from an altered innervation of the uterine vessels, dependent upon hysteria. Likewise, in a perfectly normal state of the genitals, profuse hemorrhages take place from them, which can only be explained by an abnormal state of congestion, arising from altered innervation.

In cases of amenorrhœa of the form last mentioned, there occur collateral determinations of blood to other organs, and hemorrhages may take place which are to be regarded as *vicarious for the deficient menstruation.* Such hemorrhages have especially been seen to occur from the mucous membrane of the nose and throat, also from the stomach and lungs, and in rare cases from the most widely differing portions of the cutaneous surface, without its being possible to determine other changes in these parts than those which are connected with any hemorrhage.

All these bleedings, however, also occur in the hysterical independently of the menstrual period, and without the menstruation itself being at all disordered. One can then only conclude that one has to do with local congestion of the affected organs, due to abnormal innervation.

Hysterical hæmatemesis only occurs in isolated cases. But in such one occasionally sees it take place very copiously, and with frequent recurrence, sometimes daily, or every two days, for a long time, and under certain circumstances producing highly exhausted conditions. The vomited blood is, according to the time it has been in the stomach, sometimes little altered, and at other times appears as dark masses of clots, or in the well-known form of a material resembling coffee-grounds. It is also found more or less abundantly in the stools. In two cases which I have myself seen, pains in the stomach, and a feeling of weight or fullness in the epigastrium, usually preceded the vomiting of blood for some time; after the vomiting, the patients felt relieved, and remained for some time free from pain. In other cases, as they have recently been described by Ferran, severe fits preceded the vomiting, and the stomach-ache reached an unbearable degree, so that the patients were much distressed and discomforted; giddiness and noises in the ears were associated with this, and the patients fell into a swoon-like condition. After a few minutes they again recovered, and then for the first time blood was ejected from the stomach, with violent retching.

The discrimination of these hysterical hæmatemeses from such as arise from gastric ulcers in the hysterical is, moreover, extremely difficult, and one can, as a rule, only be certain after prolonged observation. The slight influence upon the general state of the patient, the absence of gastric disturbances in the intervals, and the close connection with the fluctuations of other nervous disorders, are usually characteristic of hysterical hemorrhages. But there are cases in which all these landmarks fail. The appetite and general condition are much depressed, and other hysterical phenomena are less pronounced. In these cases, the final issue, sudden arrest of the bleeding and its renewal by other hysterical symptoms, often first affords certainty.

What has been said of hæmatemeses, in great measure applies to *hæmoptyses* in the hysterical; as purely hysterical symptoms, they occur perhaps even more rarely than the former. Again, hemorrhages of both kinds are frequently simulated; to render themselves interesting, patients produce blood, which may have come from any other source, as vomited or expectorated, or they drink the blood of animals and then irritate themselves, so as to vomit artificially. One must therefore be constantly on one's guard against deception. This remark is still more applicable to the blood-staining of the sweat and tears sometimes observed, and especially to those greater hemorrhages from particular, circumscribed portions of the skin. The latter are said to occur particularly on the hands and feet, and on the chest and forehead, and in such a manner that a bladder-like elevation of the epidermis first takes place, when the serum in the bulla, at first clear, becomes of a bloody color, and then, after flattening of the skin, a flow of blood takes place, often tolerably abundant, and persisting for a length of time. This phenomenon, described as *stigmatization*, in which superstition beholds a recurrence of the wounds and bleeding of Christ, has in most known cases become so suspicious, from the conduct of the patients themselves, and from that of the priesthood, who have derived great benefit therefrom, that it may be doubted whether we have not as a rule to deal with mere fraud. At any rate, those cases are not convincing in which a rigorous inspection has been avoided. This also applies to various cases which have been related in more ancient literature, in which not only is blood said to have issued from all possible parts, but also urine and fæces, sometimes from the umbilicus, and at other times from the eyes, ears, or any other part whatever. On the other hand, the possibility must not be denied that, under certain circumstances, in consequence of rupture of the smaller vessels, bloody admixtures may appear in the sweat and tears, and that thus also more considerable hemorrhages may occur. This seems to have been the case in a patient observed by Parrot, in whom, at different times during general convulsive attacks, an escape of bloody-colored fluid was noticed from the skin of the fingers, the knees, the thigh, the chest, and also from the conjunctiva. In other cases, again, the occurrence of an extravasation of blood into and under the skin has been noted, on the solution of the continuity of which bleeding followed; this was so in a case of Astley Cooper's, quoted by Laycock,[1] in which there was bleeding from the breast, and likewise in a case of bleeding from the scalp related by Magnus Huss.

Other Anomalies of Secretion and Excretion.

Salivation sometimes occurs with great intensity after hysterical fits, and more rarely it is met with as an independent symptom, occurring daily apart from fits. In some cases also, as Valentiner correctly points out, the salivation is only apparent, and arises from the circumstance that, on account of spasm or paralysis in the pharynx, the saliva is not swallowed, and therefore escapes from the mouth. The opposite condition, too, of *abnormal dryness of the mouth*, with a parched and burning sensation, is found in some hysterical patients, and causes them to drink incredible quantities of fluid.

Gastric Secretion.—With the spasmodic states of the stomach, mention has already been made of hysterical vomiting (not bloody). It has yet to be remarked that with this a considerable increase of the gastric secretion may frequently be determined, as large quantities of fluid are ejected, even in a perfectly fasting condition. The appetite is thereby generally much diminished. Sometimes, on the contrary, the patients are very hungry, and take all possible nutriment, although they are aware that they cannot retain it. It is a characteristic feature that occasionally particular, and indeed very substantial, articles of diet may be borne without vomiting. Thus it is not rarely possible to put a stop to it by allowing the patient to eat perfectly raw bacon or raw minced beef, with whole peppercorns.

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[1] l. c., p. 214. "In a young lady, seventeen years of age, the breast appears swollen. There are one large and several smaller extravasations of blood, resembling those due to leech-bites. This escape of blood, from which she has repeatedly suffered, begins about a week before, and ceases a week after menstruation." Laycock himself also mentions bleeding from the nipple.

In some cases hysterical vomiting is the result of a vicarious act of the stomach; that is, it has been repeatedly observed when the secretion of the kidneys is either lessened or altogether suppressed. In a case of the latter description Charcot succeeded in demonstrating urea in the vomited matter. Fernet also determined the same in one case. In a case of the same kind which I noted last year, this could not be proved. But here the suppression of urine was never so complete as in Charcot's case, as from 100 to 200 c. cms. of urine were daily voided.

As regards the abnormal accumulation of gas in the stomach and bowel, the reader is referred to what has been said regarding paralysis of these parts. The obstinate constipation of frequent occurrence has also there been discussed. It has yet to be mentioned that in other patients there exists a great tendency to profuse watery diarrhœa, which is unmistakably related to the other nervous symptoms, and may arise especially after mental emotion, just as suddenly as it occasionally disappears. It produces exhaustion much less frequently than copious vomiting does.

Abnormalities of urinary secretion are very frequent in the hysterical. A decided increase, as well as diminution of urine occurs. The former takes place temporarily, especially after convulsive attacks; under such circumstances a very copious, clear urine, of low specific gravity, is often (not always) secreted (*urina spastica*). Diminution of urine—true ischuria—is usually a more persistent symptom, which is often coupled with spasmodic closure of the neck of the bladder, so that the small quantity of urine which exists must be removed by the catheter. Charcot, whose observation has already been mentioned, has proved that, in the small quantities which are still excreted, a relatively large quantity of urea is present. I can also state the same of the case I have examined. As Charcot therefore says, it cannot be a question of diminished secretion in consequence of spasmodic closure of the ureters, as the quantity of urea must then also be relatively diminished ; but the cause of the diminished secretion is evidently to be sought in the kidneys themselves, and indeed probably in their vascular system. The possibility of tolerating such conditions is afforded by the excretion of urea by other organs. This has already been proved by Charcot in the case of the mucous membrane of the stomach; but it is probable that in other cases the mucous membrane of the bowel, as well as the skin, can act in a similar manner, just as this is observed in contracted kidney, acute nephritis, and so on.

Increase of the uterine and vaginal secretion is undoubtedly of so frequent occurrence in the hysterical because the chronic diseases of the genital organs, as a consequence of which fluor albus appears, are particularly often met with in such cases. But there are also alterations of this secretion which must be attributed to nervous influences. Thus, immediately after hysterical attacks, a decided increase of an existing leucorrhœa has often been seen to occur. This may also, however, take place periodically with a perfectly normal mucous membrane, and be dependent upon the state of the nervous system generally.

A governess was under treatment in the Lunatic Clinique at Würzburg, who at the age of forty years suffered from hysterical symptoms of various kinds, especially from globus, paroxysmal tympanites, and variable hyperæsthesiæ, and neuralgiæ, and whose disposition was decidedly depressed, though at times very lively, and particularly erotic. In this case, for several months, simultaneously with the tympanites, which usually developed itself in the forenoon, there appeared a very profuse, thin fluor albus, which had its source partly in the uterus and partly in the vagina, without its being possible, however, to recognize with the speculum any abnormal condition of the uterine or vaginal mucous membrane, beyond the moist state of their surfaces. The fluor only appeared periodically, but then frequently in such a manner, that within a short time the patient's linen was soaked; repeatedly it completely disappeared for a day, and in consequence of emotional excitement a great increase could always be determined. Local treatment with alum had no decided effect upon the symptom; but along with the tympanites it quickly and completely disappeared when the patient was put into a lively state of excitement by the visit of her sister, who overwhelmed her with reproaches.

Briquet found an *increased secretion of milk*, taking place at any unwonted period, in an hysterical patient. The secretion had begun in her case during her first pregnancy, and continued for years afterwards, uninterrupted or diminished by renewed pregnancy. The sensitiveness of the breast was at the same time so great that the patient could not suckle her children.

Hysterical Attacks.

Many of the forms of convulsion already described, when they occur alone and transitorily, may be designated hysterical attacks. In a restricted sense one understands by the latter those convulsions of a generally distributed clonic and tonic character with which peculiar psychical symptoms are very frequently united,—or even the transitory appearance of these psychical disturbances alone, without convulsions.—Such attacks sometimes take place without a demonstrable cause; in other cases they are the result of over-excitement of the most varied description ; still more frequently they arise from trivial psychical excitation. The consciousness of being under observation, and the desire to attract attention, may cause an increase and exaggeration of this, as of all other hysterical symptoms. In many patients an increased tendency to attacks is shown at the menstrual periods. At the commencement of the latter a sensation, analogous to the epileptic aura, is present, frequently in the form of the globus ascendens; or it is ushered in by a painful dragging in the extremities, or pain and giddiness in the head, or singing in the ears and darkening of the field of vision. Or, again, the patients feel generally unwell and out of humor, are in a high degree irritable, and are then so excited, by the slightest cause, that the attack takes place. In *milder cases* this consists of general rhythmical clonic convulsions of the extremities and head. Respiration is at the same time much quickened and exaggerated; occasionally a temporary spasmodic interruption or irregular action of the respiration occurs. Consciousness is not lost therewith. The patients can still dispose of themselves or lie so as to avoid injury; they hear what is said in their neighborhood, are roused by more powerful physical irritants; and it is sometimes possible, either by such, or by means of a powerful moral impression, suddenly to put an end to the attack. A single attack rarely lasts more than a few minutes; sometimes, however, a series of these follows almost immediately one after another.

1. Of the *more severe hysterical attacks*, one form is met with which cannot be distinguished from epilepsy. Consciousness is completely lost; the convulsions have alternately a tonic and clonic character; the respiration is extremely slow and stertorous; the tongue, too, participates in the spasm; foam issues from the mouth, and after the attack the tongue and lips are occasionally seen to be bitten. Such attacks are called *hystero-epileptic*. Quite the same exciting condition in the brain evidently constitutes their basis, as in the so-called "true" epileptic fits. The condition here only appears as a symptom of hysteria, while in other cases it is produced by other causes, the most varied.

2. The more severe hysterical attacks may assume yet more complicated forms, which, by some at least, are still considered as belonging to the category of hystero-epilepsy. Thus, in particular, tetanic symptoms occur; the highest degree of opisthotonos may develop itself; the patients are bent forward like a bow, merely resting on the ground with head and heels; or other distortions of the trunk and various peculiar contortions of the limbs occur. The patients then often statuesquely maintain for some time such a forced position, then the mildest movements and distortions, following one another rapidly, again make their appearance. The most remarkable positions are imparted to the extremities: the legs are thrown into the air, or contracted in extreme flexion, and again extended in violent treading movements, then forcibly crossed, or the feet approached to the face. Patients swing their arms in the air, close their fists, beat with them upon the ground or their own bodies, tear their hair, and then again, with fingers outspread in extreme extension, perform the most peculiar gestures. The countenance is distorted with grimaces, sometimes assumes a jovial grin, and again an irate, wrathful expression. Then the whole body is in commotion. The patients roll violently round their long axis, or cast themselves with force, sometimes forward and sometimes backward on to the ground, or strike their heads forcibly against

walls, so that it is only with great care that they are preserved from injury. Loud screams, singing, ejaculation of particular words, and fits of laughing and weeping, may appear simultaneously or alternately with other symptoms. The duration of these attacks varies from a few minutes to several hours; with slight intermission they are sometimes seen to continue for several days. Consciousness is the while apparently always confused, although it is not rarely possible to induce reaction by powerful irritants, and sometimes thus suddenly to terminate the attacks. Usually hallucinations and delirium are present, which, in part at least, appear to be the cause of the peculiar movements of patients. Frequently also delirium, as a so-called "psychical aura," constitutes the commencement of a regular convulsive attack. (Consult on this the section on Hysterical Mental Disturbances.) After the termination of the more complicated, as of the simpler attacks, there either follows a sudden transition to the normal state, when merely lassitude is complained of, or the patients fall forthwith into a deep sleep for several hours, from which they awake without a distinct remembrance of what has occurred, or confusion and delirium still continue for some time.

3. Another variety of convulsive attack in the hysterical, which is closely related to the last described, and may combine with or proceed from it, is the *cataleptic*. Sometimes the condition of cataleptic rigidity appears limited to particular limbs; it is frequently met with spread over the whole body, and is combined with absolute incapacity for any spontaneous movement, generally with a diminution of reflex action. Consciousness is sometimes perfectly retained in this state, so that after its disappearance patients can state exactly what has occurred to them during the attack. Sensory comprehension may, indeed, be extraordinarily acute. On the other hand, all sensation is sometimes wanting; but hence lively hallucinations appear, and the patients exist altogether in a dreamland, without being conscious of the actual state of affairs. After the return of consciousness, recollections there anent are generally very hazy. Cataleptic fits may continue much longer than the other convulsive attacks of the hysterical. Cases are known in

which they have lasted, with slight intermission, for months. In other cases they disappear in a few hours, or appear only very transitorily in alternation with other symptoms. Moreover, from these conditions of true catalepsy[1] with waxy rigidity of the limbs, we cannot distinctly separate those other conditions, likewise occurring in fits, in which, with a quite analogous psychical behavior, the muscles are perfectly lax, the extremities when raised drop the moment they are let go; as well as those conditions in which, with every attempt at passive movement, contraction takes place in the antagonistic muscles, and thereby motion is rendered difficult or impeded.

This irregular conduct is often seen simultaneously in different groups of muscles; or in the same attack the waxy flexibility alternates with both the other conditions. In cases of complete relaxation of the muscular system, it happens that the respiration becomes extremely weak, and the action of the heart and of the pulse is scarcely any longer to be felt. These are the cases which have been described as "*hysterical trance*," and in which also a superficial examination or insufficient knowledge has occasionally led to the assumption of actual death.

Finally, as regards the attacks of *somnambulism*, *sleep-walking*, *wakefulness*, *magnetic sleep*, *hypnotism*, and *ecstasy*, and whatever else those attacks have been named which are met with in the hysterical, they are merely varieties of the form last described. We have always in such cases to deal with dreamy conditions. When thus affected, patients can, actuated by the promptings of their dream, execute various complicated movements, when, like animals whose brains have been removed, without noting their surroundings, they are guided by stimuli affecting the senses, cleverly avoid obstructions, and under the most dangerous circumstances preserve their corporeal equilibrium. Sometimes they answer particular questions addressed to them in a perfectly rational manner: in these answers the influence of the revelations of their dream is frequently to be recognized. Moreover, as the sensory apprehension, as already mentioned, is occasionally quickened, so apparently wonderful powers of distinguishing objects and persons by means of the different senses are observed. One must, however, be very sceptical in testing such statements. For, on the one hand, there frequently exists in the patients themselves, who suffer from such attacks, a tendency to exaggeration and deceit; and, on the other hand, they are easily misled by their friends, who are either themselves superstitious, or trade upon the superstition of others. The peculiar conditions are then ascribed to a supernatural or divine influence, and in order to render the pretended wonder still more wonderful, it is embellished with all manner of falsehood.[1]

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[1] The term catalepsy is used in a very varied sense; by us, as a rule, only for the state of flexibilitas cerea; sometimes for all the above described conditions of loss of voluntary motion without complete loss of consciousness; by some English authors also for the more complicated attacks of hysteria mentioned above under 2, in which convulsive movements are executed with apparent premeditation.

It has finally to be mentioned that these various conditions of catalepsy, ecstasy, and so on, may sometimes occur spontaneously, without a known cause, or in consequence of powerful emotions, but also that it is sometimes possible (and indeed to the exclusion of all deception) to induce them artificially. As is known, this may be accomplished in perfectly healthy people, but, nevertheless, more frequently in the nervous, and especially in hysterical people, by various manipulations. It appears to be a point in all contrivances undertaken for this purpose to fix the attention on one object, or to direct the attention very steadfastly in one direction. The condition is most frequently induced by the subjects directing their eyes fixedly upon a dazzling object; sometimes it may be called forth by one placing the fingers for some time upon the eyeballs, or by spreading a cloth over the head of the patient. It may sometimes be produced by passing the hand over the skin, or by various mysterious gestures, which arrest the attention of the patients, or finally, by the determination of the latter to subjugate themselves to certain ideas. In some animals also, as is well known, one may induce quite similar conditions by keeping their bodies fixed for some time, or by holding dazzling objects close to their eyes.[1]

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[1] Consult, for example, the abundant literature which has already appeared in Belgium concerning that most recent hysterical wonder, Louise Lateau. The most noteworthy production on this subject is that of the Belgian psychological physician, Lefebure, to wit: L. Lateau de bois d'Haine, sa vie, ses extases, ses stigmates. Louvain, 1870.

Psychical Behavior. Hysterical Mental Disturbances.

Various psychical peculiarities and disorders of the hysterical have already been mentioned in the preceding pages. A comprehensive description of these extremely variable symptoms, and a statement of the relations existing between hysteria and other mental disturbances, is still necessary.

In the milder and more frequent cases, exalted mental irritability is the most prominent psychical symptom. Pleasant as well as unpleasant emotions are provoked with unwonted ease. Patients are timid, easily overcome by any unexpected occurrence, sentimental and sensitive. Every trifle annoys and upsets them, and there is this peculiarity, that a more recent stimulus may often effect a diversion in an exactly opposite direction. A despondent disposition, in which everything annoys and painfully affects the patients, and in which they feel miserable and incapable of doing anything, may immediately succeed an equally lively, frequently exuberant and frolicsome tone, in which they see all things in a rosy light, and are often capable of extraordinary exertion. Peevishness is the leading characteristic of the hysterical; it is the expression of exaggerated psychical activity.

Nothing else is possible than that, under such circumstances, unpleasant emotions should on the whole preponderate, as excessive activity always has a tendency in that direction. An unamiable disposition is therefore as a rule pronounced in the hysterical, is a source of grief to the patients themselves, and renders them objects of solicitude and anxiety to their friends. In more matured cases the condition is more painful, melancholy alone prevailing, and not rarely continuing for years, and any change of disposition which even then does not fail to occur is yet only partial, more endurable periods sometimes interrupting those which are altogether grievous. In these states of ill-humor, pains felt in different parts of the body always play a distinguished part. These, as we have seen, are partly the immediate consequence of exaggerated mental activity, and to some extent arise independently in consequence of increased irritability in particular sensory tracts. In both cases they serve to nourish the existing diseased tendency, and promote and maintain the same, just as conversely an exaggeration of abnormal sensations and involuntary movements is caused by the attention with which their physical state is noted by the patients. It will, however, be evident hereafter that abnormal sensations alone cannot by any means generally be regarded as the cause of hysterical ill-humor, any more than this can occur in the purely hypochondriacal tendency, which is met with unconnected with hysterical symptoms.

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[1] This has been longest known in the case of fowl, which may best be "hypnotised" by holding their bodies gently but firmly on a table, while with the other hand the head is directed to the same. A chalk line drawn on the table past the beak favors the development of the phenomena. Czermak has recently demonstrated the same in other birds. It is, moreover, very easy, as the same authority has shown, to call forth this phenomenon in crabs, which it is only necessary to hold between the fingers for some time to see them remain motionless in the most forced attitudes. A similar condition may not rarely be observed in rabbits and guinea-pigs.

The exaggerated psychical irritability of the hysterical usually finds expression also in the appearance of various idiosyncrasies and peculiar inclinations. Some hysterical people, in the presence of, or by reflecting upon, particular and altogether indifferent objects, experience the highest degree of repugnance and distaste. Especially often does the aversion to certain animals exist, such as frogs, mice, spiders, cats, and so on, whose presence frequently calls forth regular paroxysms of excitement, convulsions, and fainting. Others, on the contrary, render themselves conspicuous by the excess of their apparent partiality for particular animals, cats, dogs, birds, and the like. In their intercourse with their fellow-creatures, too, they frequently show themselves to be ruled by the most violent and altogether unfounded antipathies and sympathies. Hatred and love for particular individuals occur in unwonted measure and without definite motive, and are displayed, now toward one now toward another, according to the accidental changes of disposition.

The sexual processes which so frequently play a part in the causation of hysteria quite often leave their impress upon the psychical picture. Although it is absolutely incorrect to deduce hysteria exclusively from insufficient sexual satisfaction, as was formerly done, and to consider all hysterical women as amorous and nymphomaniacal, yet it is not to be denied that the imagination of many such takes an erotic direction, and that in some cases sexual sensations prevail. It may be determined with tolerable frequency, in the case of exquisitely hysterical maidens and women, that an exaggeration of symptoms occurs in the presence of men. Some ailments evidently owe their origin simply to the desire to be physically examined by the physician, or at least to retain him as long as possible in their presence. As a rule, therefore, those physicians are preferred by the hysterical who lend a ready ear to all their complaints, and trouble themselves most with them. Such men, however, by no means always exercise the most favorable influence upon the diseased condition.

But it is not only to the physician and in consequence of sexual excitement that the hysterical are troublesome and prone to exaggerate their symptoms. Their entire disposition causes them to be burdensome to their friends, and chiefly in this way: the feeling that they are destined to suffer more than others on account of their unfortunately nervous constitution renders them selfish, and regardless of those other "rougher" natures. They wish it to be understood, not only that they are ill, but that they have to suffer in an altogether unprecedented manner. Frequently it is much less of their bodily ailments that they complain than of their life's destiny, which they consider a very hard one. One very often meets with this kind of exaggeration in elderly hysterical females, in whom, in fact, external circumstances have furnished elements calculated to embitter and to develop the hysterical condition, whether it be that they have not attained the longed-for goal of matrimony, or that they have not found in marriage corresponding physical and moral satisfaction, or whether it be that as widows they have to endure grief and want, or finally, that they must needs part with their grown-up and now independent children, and regard their lives as objectless and desolate. They frequently assume the mask of resignation and quiet endurance; but although they protest against any attention, and ever maintain that they are not worthy of regard, yet are they annoyed and made ill by every trifle, and demand the most constant solicitude from their friends, whom they continually torment by their resigned air and indirect reproaches.

But the same thing is also met with in every degree in younger hysterical girls and women, although, upon the whole, their exaggerations have more frequently reference to symptoms of disease. Some, in order to arouse attention and consideration, injure and burn themselves, maintain purulent cutaneous sores by using irritating ointments for a length of time, swallow needles, or stick these under the skin in the most varied situations, or attempt to hang, drown, or poison themselves, and so forth. Their arrangements are, as a rule, so made in such cases that the attempt is noticed and frustrated in time by their friends; but real attempts at suicide are also met with, or accidentally such a design proves fatal, even against the will of the patient. Others try to attain the same desired end by direct falsehood and deceit, which is sometimes carried out with such cunning as to cheat for long their friends and physicians, but they are as a rule easily detected.

Again, it has happened that the hysterical have drank urine and eaten even excrement, to vomit the same, and give rise to the belief that the said substances proceed from their stomach. As regards cutaneous hemorrhages which are frequently seen in the hysterical, it has already been stated that they only arise spontaneously in rare cases, and are as a rule artificially produced by deceivers. Some hysterical people would lead one to believe that urine issued from their navels, breasts, ears, eyes, and so on (a phenomenon which, under the name of paruria erratica, was of yore generally recognized as a symptom of hysteria). Others have introduced living or dead animals (toads, frogs, worms, etc.) into the anus or genital organs, and in the presence of a credulous public have reproduced them. Yet others pretend to have had no evacuations from their bowels for months or years, to have existed without food, or to have lived upon consecrated wafers, and so on.[1]

In other cases the tendency to lie and cheat takes yet more objectionable forms; the patients intrigue against their friends, state that they are the victims of the animosity of the latter, maintain that they have been persecuted and outraged, and sometimes play their part so well that not only the general public, but also physicians and judges are deceived.

Another symptom which sometimes appears very prominently in severer forms of hysteria affords the clue to understanding some of these occurrences. This is the deep-rooted and persistently painful tendency, unbroken by any pleasant emotion, to "uncontrollable impulses" (Zwangvorstellungen). The patients frequently can only affirm that they feel themselves driven to do something bad to procure peace; sometimes it is particular acts which float before their imagination, and the thought of which pursues them continuously until they are carried out. Occasionally they are compelled to mutilate themselves, and again to injure others. Sometimes they succeed by loud abuse and cries to avoid their impulse; at other times they rave, sing, dance, make a noise, and destroy everything they can lay hands upon, with the full consciousness and in the belief that they may thus gain relief. At other times, finally, it is the thought that their friends are anxious to render them uncomfortable by wicked and underhand devices, which pursues them in like manner, and occasions falsehood and deceit.

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[1] Although medical works in which such marvels are credulously referred to have happily become rare (in the older writings they naturally play a great part), yet such are not altogether wanting even at the present day. The pamphlet of Lefebure on Louise Lateau, the stigmatised and wafer-nourished, has been already mentioned. Of others undertaken in the same spirit, there is one, published in 1858 by Dr. I. Ch. Seitz, of Pest, viz.: The history of a rare and incomprehensible disease, etc., with an appendix on frogbirths. We are therein told of a young girl from whose eyes fruit pips sprang, from whose ears and navel fæces escaped, and from whose anus and genitals fleshy shreds came away, while worms with black eyes were vomited, and many other such things; further, of a woman from whose genitals four and twenty living and dead frogs were passed, some of these indeed with cords of attachment. The birth of the frogs was witnessed and believed in by several physicians.

It would be very unjust to conclude from the frequent tendency to deceive met with in the hysterical, that most of their symptoms, including convulsions, paralysis, and so on, were, as a rule, intentionally produced and simulated. But we can well understand that many of the symptoms which awaken such a thought are the involuntary results of the lively and peculiarly powerful imagination which is coupled with the existing irritability of the patients.

This is seen most distinctly in those cases in which hysterical symptoms are transmitted from one individual to another by means of psychical infection. This occurs partly in individual sporadic cases and partly more extensively in actual epidemics. It is generally the hysterical fits of different kinds (the most complicated forms of convulsions as well as the states of catalepsy, ecstasy, and the like) which so multiply; but paralyses, anæsthesiæ, and hyperæsthesiæ may also similarly infect. As has been already stated, it is always individuals already predisposed upon whom this infection is operative, and in more extensive epidemics universally active predisposing causes may be demonstrated. But the repetition of particular symptoms of a peculiar kind in almost every epidemic is only to be explained by supposing that the powerful excitement caused by the representation of certain sensory and motor phenomena brings about the actual occurrence of the latter.

Not less distinctly is the influence of the imagination seen in a converse manner, when it ameliorates or abolishes hysterical symptoms. The conviction awakened in the patient, by what means soever, of the possibility of cure, may almost suddenly abolish apparently the most severe and obstinate symptoms of disease, which may have existed for many years. It is sometimes possible for the physician or for other people to influence the patient, and by their conversation to create such a conviction. In other cases the efficacy of particular remedies becomes an article of faith with the patient, and by this faith are they cured, equally whether we have to do with medical or with any other remedies. All possible quack ointments, homeopathic, sympathetic and religious influences may, under certain circumstances, have the same effect, if it be only possible to create a sufficiently firm conviction in the patient. Threats are likewise effectual in some cases, and especially during the epidemic prevalence of the disease. From all that has been said, it immediately follows that the power and energy of the will undergo many alterations in the hysterical. Generally speaking, it may be said, that in a ratio corresponding with the exalted sensitiveness of the patient, impulses of the will increase in frequency and power, but that the frequent change of temper compromises the consequences of this. The eccentricity of many of the hysterical is shown in never-ending undertakings and their abandonment, and in spasmodic and imperfect work, without requisite energy. But in other cases one finds in them so great an endurance and resolution of will as is scarcely to be found in less sensitive people. Testimony is borne to a one-sided energy of will in those cases, in which they subject themselves to the most excruciating physical pain, and endure the same with stoical indifference, merely to render themselves objects of interest, or in which they perpetrate the most complicated frauds, with a like intention. Moreover, in useful matters, *e. g.*, in nursing, in activity in benevolent societies, and so forth, an incredible amount of endurance and self-sacrifice is exhibited by the hysterical. If it is only possible to arouse their enthusiasm for any object, or to awaken their emulation, it may happen that patients who have hitherto lived in the greatest indolence and self-indulgence, and who have appeared incapable of executing the slightest work continuously, suddenly undergo the greatest exertions and are roused to an untiring and persistent activity.

But more frequently the power of will is small. At most the patients rouse themselves to transitory and limited endeavors; on the whole, they are the apathetic prey of their whims and fancies, which, through this very failure of will energy, take the upper hand in all their dealings. Even the wish to make up their mind upon necessary matters causes a painful sensation, and the slightest exertion seems an insurmountable barrier.

In some cases this abulia reaches such a degree that the patients, when perfectly conscious, abandon almost every movement, do not leave their bed, take no nourishment, and sometimes even do not observe the most necessary cleanliness. It is only when one attempts forcibly to drag them from their couch, that they prove, by the energetic resistance called into action, that they have not altogether lost the power of motion.

Hysterical mental disturbance.—By this term one understands those conditions in which the psychical symptoms of hysteria described are present in a more pronounced form and persist for a longer time. Strictly speaking, the simpler attacks of hysteria may also be considered psychoses, and in any case the limit to which the term mentioned has been pushed is quite undefined. The degree of disturbance, as a rule, decides the point less than its duration. Grave disorders of consciousness occur in connection with hysterical attacks, such as hallucinations with erroneous ideas, dreamlike conditions, transitory outbursts of mania, and so on. Sometimes the entire hysterical paroxysm is constituted simply by such psychical phenomena. But these temporary disturbances, which are soon succeeded by a normal state, are generally described merely as deliria, while one finds the term hysterical mental disturbance only applied when persistent psychical changes exist. Intermediate conditions are, however, numerous. It is rarely that in patients who suffer from complicated attacks the psychical behavior is in the intervals perfectly normal. Some of the lively images which appear during such paroxysms are subsequently considered real. If the deliria are frequently repeated, and these, as a rule, take much the same form, a few false ideas gradually remain; later, more complicated insane creations are developed.

The subject of the delirium varies with the former mental condition of the patient. It is frequently religious; the sufferers see heaven open and have divine visions, converse with the saints, and hear and promulgate prophecies in prose and verse. In like manner do the attacks of "being possessed" (Besessensein) arise, which have their origin in religious discourses, and which played a great part in the hysterical epidemics, especially of former centuries, but also at the present day are not altogether rare. Patients think they can recognize the devil or some other wicked spirit in their interior, feel that they are urged to physical strains and cramps by the same, and are compelled by these unclean agents, in spite of themselves, to utter unholy words, blaspheme God, laugh at the priest, and so on. Others talk in a different key, according as one or another spirit urges them to speech. Quite analogous, but less known to the superstitious, and therefore less used by such, are the cases in which patients feel themselves magnetized, electrified, or physically acted upon in some other indefinite manner, and refer their convulsions and peculiar contortions to these physical agents.

In other cases patients are transported to fairyland, where milk and honey flow, and are overwhelmed with power and riches, and where, finally, the Prince is not wanting who raises them to his throne. Above all, an erotic character is rarely absent in these deliria; it appears as well in the visions of heavenly brides as in the declarations of the possessed of Satan, and as in those influenced by magnetism or otherwise. It is seen most pronounced in those cases in which patients erroneously suppose that they have been ravished by those who have access to their person. And in such cases details are sometimes recounted with so much minuteness, and deviate so little from what is possible, that on several occasions trials have been instituted upon such accusations, and in a few instances convictions have followed.

If such attacks of transitory psychical disturbance only rarely appear, their impression is gradually obliterated, and the remaining mental life of the patient is not further influenced. If they multiply, however, persistent mental aberration is more and more induced. But patients are even then sufficiently wide awake for a long time, and conceal their mad ideas, the continuance of which is only to be recognized by their ecstatic and fanciful demeanor, or by their peculiar, reserved, irritable and irascible behavior. Hallucinations sometimes cause them, even in the intervals of attacks, to betray it. The delirium easily breaks out in consequence of external causes and excitations; but much more frequently does one find that it makes its appearance in a perfectly periodical manner and without demonstrable cause, being regularly introduced by a condition of greater irritability and ill-humor.

In other cases there arise continuous psychical disturbances, which are not particularly affected by the fits, although certain typical variations in the intensity of the symptoms always exist.

The character of the affection in a portion of these cases is merely that of *melancholy*, and that, too, in its purest and simplest form. Without ever having hallucinations or mad ideas the patients feel constantly wretched, anxious, incapable of enjoyment in the society of others, complain of a miserable sense of desolation and a painful inability to make up their minds or work. Under these circumstances those uncontrollable impulses before mentioned usually appear, and urge them to self-mutilation or attempts at suicide. It is just in these cases, too, that one frequently finds the desire to intrigue against, slander, and injure others, thereby to derive a certain satisfaction for their own ailments. Hence, these patients cause much unpleasantness both in the family and in asylums for the insane, in which they have frequently to be confined.

This is still more true of the second form of continuous hysterical mental disturbance, the phenomena of which correspond, as a rule, to the clinical picture of so-called *folie raisonnante*. This almost always affects patients in whom the tendency, at least, to hysteria is congenital, although occasionally symptoms of it first appear at a later period. Such patients are completely ruled and hindered in their work by their ever-changing fancies; though sane enough to know that thereby they come into conflict with their surroundings, they are unable to curb their inclinations and impulses; the moral sense that they should strive against this is either originally absent or is lost in consequence of the disease. For any useful or steady employment they are unfit, but pursue their own selfish aims with the greatest perseverance; the sexual appetite is often strongly developed in them, and indulged in a shameless manner; not rarely they are given to drunkenness. The desire to multiply vices is the predominant feature in their endeavor; in the most adroit fashion they cheat, lie, and steal, and in the latter case, usually, see the necessity of casting suspicion upon the innocent. They frequently go to work with much spirit and cunning, and ever seek to cloak or palliate the perversity of their actions by clever argument. An overweening egotism at the same time characterizes all they say; without laboring under actually erroneous ideas of their position, yet they always have a sense of superiority, and imagine all must make way for them. The weakness present in all the ingenuity, moreover, reveals itself in these as in cases of folie raisonnante arising from other causes, in the never-failing belief in the incontrovertible nature of the arguments adduced. But many of their falsehoods and deceptions are usually the expression of deeper disturbances of intelligence. They themselves believe what they say, their memory deceives them, and reproduces previously related falsehood in the sense of their ruling passion. Sometimes they also suffer from hallucinations which may gradually transform themselves into completely established erroneous ideas. Occasional more powerfully excited conditions are rarely absent, and are produced either by conflict with their friends or arise spontaneously. Confusion, delirium, loss of ideas, and pronounced *maniacal states* are thus developed.

Finally, *primary derangement* (primäre Verrücktheit), which may develop, partly with and partly without hallucinations, may be described as a third form of chronic hysterical mental disturbance. These cases are closely allied to those arising from paroxysmal delirium, of which we have already spoken. The delusion of being possessed, magnetized, or otherwise haunted, and ideas of importance (Grössenvorstellungen) of the kind described, may gradually develop without the assistance of fits. Here, too, sexual ideas are not, as a rule, absent. Conditions of this kind always advance progressively, and gradually lead to ever greater fixation and increase of erroneous notions, and finally, to imbecility. In other forms of hysterical mental disturbance, also, this is usually the ultimate result; yet they may retain a considerable amount of intelligence much longer.

Progress and Results.

Hysteria is almost always a chronic disease, which exists for many years, and, as a rule, only completely disappears at an advanced age. Even cases commencing suddenly, in which a violent succession of severe symptoms is frequently observed, rarely recover quickly; usually, after a comparatively short time, a remission occurs; but distinct hysterical symptoms continue, which on every opportunity may become aggravated.

To determine exact stages in the progress of hysteria is impossible, on account of the great variability of individual cases. In cases with a powerfully developed predisposition the increased irritability and peevishness is well marked, even in childhood; the convulsive attacks generally first occur later, at puberty. On the whole, they are met with at this period most frequently, and often completely disappear, after its termination, without the disease on that account ceasing to exist. On the contrary, it is just then that other, and especially the psychical phenomena, appear more prominently and persist.

The intensity of the psychical phenomena, moreover, stands in no constant relation to that of the other symptoms. There are hysterical persons, in whom the peculiarity of the psychical behavior is only evident in a small degree, while paralyses, pains, anæsthesiæ, and so forth, are powerfully developed, and frequently remain obstinately fixed for a long time at particular parts of the body (so-called local hysteria), while in other cases the psychical symptoms come to the front, and there are merely indications of the rest.

Numerous changes almost always occur in the course of hysteria, both as regards the combination of particular symptoms and their intensity. Exacerbations often occur regularly at the menstrual period; they are, moreover, induced by the most varied external influences. Remissions may likewise be thus induced; sometimes they appear without any recognizable cause, and occasionally interrupt in an altogether unexpected manner the fatal monotony of symptoms, when one has looked upon recovery as no longer possible.

These remissions are, as a rule, only transitory, and after a longer or shorter period, either the former or other hysterical symptoms again make their appearance. Many of the so-called CURES of hysteria are, in fact, nothing else than such remissions; the continuance of the disease is distinctly shown by the fact, that the slightest cause is sufficient to call forth severe symptoms again. At all events, the complete cure of hysteria before the period of involution is a rare event. It takes place most frequently in those cases in which the disease has been acquired and in which it is possible to remove the exciting injurious influences.

The higher degrees of hysterical mental disturbance, as a rule, first develop themselves after the disease has existed for a long time; these are, moreover, for the most part cases of inherited hysteria. Sometimes they first distinctly appear at the climacteric period, which is in other cases the turning-point towards recovery. It sometimes also happens that the first symptoms of hysteria appear at puberty, that the disease then remains latent to a certain extent, again to reappear in a pronounced manner at the period of involution.

We have repeatedly spoken of mild and severe cases of hysteria, and it is evident that there are very different degrees, from those simple forms of increased irritability to the rooted psychical alterations, the attacks of general convulsions, and the widespread paralyses and anæsthesiæ which one sees in other cases.

But it is scarcely possible more definitely to limit the different degrees of hysteria. If (like Dubois) we regard as a test the violence of the hysterical fits and the degree of disturbed consciousness during these, we shall form quite erroneous conclusions. There are cases with fits, very severe, but separated by long intervals, and it may happen that in these intervals scarcely any noteworthy disorders exist. In other cases fits are altogether absent, but the psychical function is so persistently abnormal, and all attempts at cure are so perfectly fruitless, that we cannot certainly regard the latter as milder than the former. One can, as a rule, best determine the amount of disturbance by the degree of persistent psychical abnormality, although this again affords no measure of the intensity of other symptoms. If fits, paralyses, anæsthesiæ, and contractions associate themselves with the severer psychical disorders, we have to deal with the most aggravated forms of the disease. On the other hand, those cases are often comparatively mild in which, by psychical infection, convulsions and other phenomena, even grave disturbances of consciousness, are called forth, the appearance of which under other circumstances would have to be considered very serious.

Hysteria has only rarely an influence upon the duration of life in the case of those affected by it. Even states of extreme marasmus, which are occasionally produced by it (hysterical vomiting, hæmorrhage, etc.), are rarely of such evil omen as when they result from other diseases. It happens more frequently that exhausted conditions, otherwise originated, which produce hysteria, cause the fatal termination. But cases are also known in which the disease alone was to blame for this, and in different ways in particular cases. Under the name of *acute fatal hysteria* cases have been described, in which, after a course of severe hysterical disorders, which, as a rule, have owed their existence to a psychical influence, death has occurred within a few days or weeks. In some of these cases numerous and powerful epileptiform convulsions appeared; as in the case of true epilepsy, a cyanotic condition developed itself, the patients collapsed, became soporose, and in this state died. Wunderlich was able in one such case, shortly before death, to determine a distinct rise of temperature to 43° C. [109.5 Fah.], without its being possible to discover a local process in the nervous system or in other organs as a cause for this. In Rullier's frequently quoted case (Thèse de Paris, 1808), epileptiform fits appeared, and also a persistent sense of constriction in the pharynx and larynx, so that a clinical picture, similar to that of hydrophobia, was given rise to. The patient in question, a girl fifteen years of age, became ill in consequence of a fright during menstruation, and after her condition had lasted for two days, she died in an epileptic fit. In her case, also, no changes were discoverable in the brain. The cases related by L. Meyer are of rather a different nature. In these, acute insane excitement of a nymphomaniacal character was first developed; general convulsions followed, as also contractions and rigidity in particular muscles; the patients quickly collapsed, and died after the disease had existed for a few weeks. Here, too, any change in the nervous system was absent. Nothing definite can be said concerning the true cause of death in such cases. It can only be surmised that the diseased processes in the nervous system itself, which occasion the delirium and the other symptoms, also ultimately cause paralysis of the respiratory muscles and heart, as in those conditions of so-called delirium acutum which are quite identical, but arise without hysterical symptoms.

But the fatal results described occur not merely in acute cases of hysteria. After a tedious and chronic course, a like violent episode may put an end to the disease and life; as a rule milder chronic disturbances have preceded for a length of time the so-called acute conditions, as, for example, is stated in the three cases related by Meyer. Also in a second case of hysteria with a fatal result, related by Wunderlich, the disease was chronic; a patient who had suffered for years from all possible hysterical symptoms, paralyses, hyperæsthesiæ, loss of sight and smell, going on even to difficulty in swallowing, and vomiting, fell into a state of marasmus, without evident physical cause, got more and more emaciated, and finally died with febrile symptoms, without the necropsy revealing changes within or without the nervous system which could be regarded as the cause of the phenomena observed.

Indirectly, in consequence of hysteria, death may take place in those cases in which patients mutilate themselves, with or without the intention of suicide. It has been already stated, that the attempts at suicide of such frequent occurrence in the hysterical are only rarely undertaken in earnest. I know several such patients, who have already attempted all possible kinds of suicide, have cut their throats, throttled, hanged, and burned themselves, leaped into water, and swallowed phosphorus and sulphuric acid, but on each occasion, under circumstances which rendered rescue probable. But cases are likewise known in which suicide has actually been effected by the hysterical, and, moreover, instances in which death resulted from self-mutilations undertaken with other intent. Thus, swallowed needles, which frequently pass through the stomach and bowels without much difficulty, have in some cases penetrated the walls of the digestive canal, and caused fatal peritonitis.

Diagnosis.

There is no difficulty in diagnosing hysteria if we have to deal with fully developed cases, and have an opportunity of watching a considerable portion of its variable career. Indeed, it is frequently possible to recognize the presence of the disease at first sight from some characteristic symptoms, above all from the psychical behavior.

In other cases, difficulties arise in various ways. In the first place, it is scarcely possible to distinguish hysteria distinctly from those conditions of so-called *nervousness*, *nervous temperament*, *increased irritability*, or whatever else it may be designated. In these states all the actual elements of hysteria exist, and their progressive development merely is necessary for the outbreak of the disease. We may therefore regard it as an hysterical tendency, or mild degree of hysteria. By taking this view we can say with Sydenham, that the majority of women are hysterical. It is, however, more convenient to maintain the distinction as much as possible, as otherwise our conception of the disease becomes extremely hazy: the tendency to the disease also does not by any means necessarily progress to the disease itself; it may remain undeveloped, and from allied influences may also favor the development of other forms of nerve disease.

From what point, then, is the term hysteria applicable? The frequent change of temper, the exaggerated sensibility to psychical impressions also occur in nervous persons; some, too, of the spasmodic symptoms appear in these, such as globus and the increased action of the heart and respiration, etc., frequently on very slight occasion. At any rate, it is questionable to make the diagnosis of hysteria dependent upon the presence or absence of a particular symptom. But its presence may be assumed with certainty, if the psychical peculiarities which mainly characterize it are distinctly pronounced and persist, if the variations of temper make their appearance without any external cause and are excessive in all directions, and if the corresponding alterations of volition and of the imaginative faculty exist. Under such circumstances, some at least of the characteristic disorders of sensibility and motion may always at the same time be demonstrated. Indications at least of attacks are present, if only in the form of occasionally occurring fits of laughing and weeping, of globus, singultus, and the like. But one must likewise number those cases with hysteria in which the fits are developed in a characteristic manner, with a less severity of the persistent psychical symptoms, whether they be the general convulsive attacks, or those of catalepsy, ecstasy or hypnotism. On closer observation it may nevertheless always be determined in such cases, that the psychical alterations, though, it may be, trivial in comparison with the severity of the fits, are yet never completely absent.

Other individual symptoms of hysteria may also appear in so characteristic a form that they can easily be recognized as hysterical, even when the psychical condition is less marked. This applies both to the different forms of paralysis, and anæsthesia, and to spasms and painful affections in particular organs. But the difficulties of diagnosis become greater the more isolated such symptoms appear, and the more obstinately they remain fixed in particular parts of the body. The point to be determined in such cases usually is, whether so-called "local hysteria" or more deeply-seated anatomical changes of the part affected are present, and it is evident that this point is of the greatest importance in reference both to prognosis and therapeutics. In describing particular symptoms, notice has already been taken of the manifold difficulties which beset this part of the differential diagnosis, and of the deplorable mistakes which may be committed with regard to this subject. It may in general be said, that the most careful investigation of the whole state of the patient in all suspected cases, is as indispensable as the minutest examination of the parts in which the local symptoms appear. The absence of visible and palpable changes in the organs which are the seat of severe pains, frequently affords a good reason for the assumption of the nervous character of the disease. But oftentimes it is only after long observation that it is possible to attain certainty as regards this, and particularly also as to whether a combination of hysteria with local disease does not exist.

With regard to the discrimination of certain cases of hysteria from cases of *actual disease of the vertebræ and spinal cord*, reference may in all essentials be made to what has been already said. Pain and sensitiveness of the vertebræ, which are so frequently met with in the hysterical, are comparatively rare phenomena in diseases of the spinal cord. The exciting causes which are wont to induce hysterical paralyses (as a rule, psychical influences) do not easily lead to organic changes in the spinal cord. But it is not to be denied that cases of the latter kind occur, as also that hysterical paralyses do not always distinctly proceed from such influences. Some cases of hysterical paraplegia, with disorders of sensibility, exalted reflex excitability, and contractions, so completely resemble others in which myelitis or sclerosis exists in particular portions of the cord, that the diagnosis is only formed from the progress of the case, and from symptoms otherwise present. It will be least possible to distinguish cases of so-called paralysis ascendens acuta, which run a favorable course, from hysterical paralyses progressing similarly. Moreover, there are some cases of *multiple sclerosis of the brain and spinal cord*, which can only with certainty be diagnosed from hysteria in their later stages, and by the final issue. These are the cases in which the paralytic phenomena frequently alter their position, paroxysmal exacerbations, and frequently as sudden ameliorations, take place, and convulsive attacks and disturbances of consciousness of a like complicated nature, as in hysteria, are met with. The disorders of deglutition and articulation, also characteristic of the former disease, are now and again observed in hysterical people. *Hysterical hemiplegia* is, as a rule, easily distinguished from paralyses caused by apoplexy and encephalitis, by the well-marked disorders of sensibility, and by the absence of facial and lingual palsy, yet in certain localized affections of the brain the same symptoms occur as in hysterical hemiplegia, and occasionally the characteristic disorders of sensibility are absent in the latter. The appearance of secondary trophic changes in the palsied extremities almost always argues the material character of the disease.

To distinguish the *convulsive attacks* of the hysterical from those of the epileptic is in some cases as easy and simple as it is in others impossible. Attacks occur in exquisitely hysterical persons which must in all respects be regarded as epileptic, and which can only be recognized as symptomatic of hysteria in that they, like the other symptoms of this disease, are changeable in their outbreak, and may be supplanted in the most variable manner by other symptoms. Their definition as hystero-epileptic can only be understood in this sense. At all events, the same exciting process in the brain constitutes the basis of the individual attack, whether it arise as the result of hysteria, or in consequence of one of those other numerous causes, which induce true epilepsy.

Under certain circumstances, the differential diagnosis between hysteria and *hypochondriasis* may be very difficult. Some have regarded these as essentially the same, and by one of these names have described both diseases, or have classed them under the general name of vapeurs, etc., or even, like Bouchut, extended the signification of general nervousness so far that the most varied cases of hysteria and hypochondriasis could be included in this term. Others finally have considered the hysteria of the female, and the hypochondriasis of the male, as phenomena of one and the same disease, which merely receives a different impression from the varying psychical constitution of the two sexes. I believe we must in the meantime maintain the distinction between the two diseases, although it is not to be denied that they present many points of alliance and pass over into one another. In cases of hysteria in which the characteristic peculiarities of the psychical conduct exist, and particularly the perpetual variations in temper, they cannot easily be confounded. Paralyses of the kind met with in the hysterical almost never occur in the hypochondriacal, convulsive attacks only much more rarely and not in the same variety as in the hysterical. On the other hand, a sort of ill-humor supervenes in some cases which can only be described as hypochondriacal. The phenomena of hypochondriasis here associate themselves with hysteria, as do in other cases the phenomena of epilepsy, or those of chorea. Conversely, the phenomena of hysteria may later appear in cases at first purely hypochondriacal. We must first possess a more exact knowledge than we at present do of the physiological connections of the symptoms of both diseases, and of the processes in the nervous system which constitute their basis, before we can more sharply establish the boundaries between them, and probably also in other important directions.

Prognosis.

The prognosis of hysteria, quoad vitam, is almost always favorable. Cases of a fatal issue of the disease (from suffocation in consequence of spasm of the glottis, or in coma after severe convulsive or cataleptic fits, or from exhaustion after conditions of intense excitement, of an acute or chronic character) are so extremely rare that one may in the case of hysterical people be unconcerned even with such symptoms as must awaken the gravest anxiety when they appear as a result of other diseases. This applies, for example, to the marasmus which sets in after persistent hysterical vomiting, or after repeated hemorrhages, as also to the profound syncopies which sometimes make their appearance in patients, with and without fits, and so on. Nevertheless, we must always remember the possibility of a fatal issue in such cases. If other physical diseases exist, in whose train the hysteria first arose, these may of course render the prognosis, as to the life of the patient, unfavorable.

But rarely as hysteria, pure and simple, causes death, so rarely does complete recovery from it take place. Even where the latter has apparently been effected, it is, as a rule, observed that the slightest cause is sufficient to reproduce the disease. In this respect, the prognosis appears most unfavorable in those cases in which the tendency to the disease is congenital, and in which symptoms of it first appear in childhood. In such cases, an exaggeration of these may usually be predicted to occur during the period of puberty, although it is true that after the termination of this period considerable remissions sometimes take place. But there likewise then occur further exacerbations, which cannot always be distinctly referred to particular exciting causes. The last hope of such patients is the age of decrepitude, but again, it is just the period of involution which frequently induces exacerbations, and gives an impetus, especially to the more inveterate and, in their prognosis, absolutely unfavorable forms of mental disturbance.

The most favorable prognosis may be given of those cases in which the tendency to hysteria, and the disease itself, have been acquired from demonstrable physical ailments amenable to treatment. A persevering treatment is most frequently rewarded, particularly when the causes are states of exhaustion resulting from severe maladies, or after great but not persistent hemorrhages; also when hysteria has been induced by affections of the genitals, the cure of which is possible, or by conditions which are in themselves of a transitory nature, as in many cases of chlorosis. If, on the other hand, the causal diseases are themselves of a chronic and incurable nature, if they do not also threaten the life of the patient, the prospects of curing the hysteria are bad. But even in cases in which the fundamental ailment can be removed, and in which there is no congenital predisposition, we must not be too sanguine in our expectations. It is very frequently observed that the altered condition of the nervous system continues, even when the cause which induced it no longer exists. It is indeed frequently possible to get rid of the more acute and grave symptoms of the disease; but the tendency to further outbreaks remains implanted in the nervous system, and on every opportunity again comes into play.

The same applies to cases of hysteria caused by powerful psychical excitement. They do not by any means always justify the favorable prognosis, which we might expect from the short duration of the injurious influence. On the other hand, the forms of the disease which arise purely from psychical infection, are sometimes rapidly cured by simply isolating the patient or by suitable moral and physical treatment.

The prognosis with respect to the intensity of particular stages of the disease differs from that regarding its progress as a whole, as also from that respecting individual symptoms of hysteria. We have already repeatedly spoken of the unexpected remissions which may occur after the long continuance of apparently grave symptoms. Such remissions take place, in cases both of congenital and acquired hysteria, sometimes without evident cause, and again in consequence of moral influences of the most varied description; at other times these remissions are to be attributed to the fact that the cure or improvement of the physical diseases constituting the basis of the hysteria follows by natural or artificial means. These remissions may under certain circumstances be so complete that it is with difficulty we can recognize the continuance of the disease, or first observe it by the reappearance at a later period of exacerbations, unexpected and without sufficient external cause. Sometimes, however, merely the predisposition to hysteria persists, and considerable exciting causes are necessary again to cause an outbreak.

The individual symptoms in the sensory and motor spheres, both alterations of circulation and those of secretion, are collectively capable of recovery, even when they have existed never so long or been repeated never so often. It cannot positively be predicted of any one of them whether it will persist or disappear; but one usually finds that, if the same symptom repeatedly shows itself, it becomes more obstinate, and later can no longer be removed by the same means as on its first appearance. It can almost never be predicted what symptoms of hysteria will yet transpire in the course of a given case, although the frequency of certain combinations, such as paralysis with anæsthesia, spasms with increased sensibility to pain, and so on, sometimes affords ground for surmise in this respect.

Deeply rooted and persistent psychical disorders afford the most unfavorable prognosis of particular symptoms. Transitory phenomena of this kind, even when they are very violent, may remain without any greater significance for the further progress of the case. But if the signs of moral perversity, of folie raisonnante, are once distinctly developed, or if fixed erroneous ideas have formed, a complete restoration is almost never to be expected. This is more likely to occur in the purely melancholic conditions of the hysterical, if these have not already played a prominent part in the active development of the disease, and thus become an ineradicable habit.

The attacks of ordinary insanity, which make their appearance in some cases of hysteria for a shorter or longer period, are likewise capable of complete recovery (Rückbildung), but are apt to induce irremediable conditions of psychical weakness.

Treatment.

1. *Prophylaxis.*

The rarity of complete cures of the disease, when it has once fully developed, renders it the more important to combat and impede its small beginnings, lest from a condition of hysterical predisposition the disease itself should be evolved. Such an influence is chiefly possible in childhood and youth; but the physician, of course, is, as a rule, consulted when the evil consequences of a defective regime come to light, and we have already to treat no longer the tendency, but the disease itself. If one has an opportunity of giving timely advice in cases of congenital predisposition, we must treat in accordance with the indications, which have already been mentioned in discussing the etiology, that is, insist upon the avoidance of those educational defects which notoriously assist the development of hysteria. As physical debility so frequently exercises an injurious influence in this respect, it must be combated by all the means at our command. Diet especially must be superintended, and we must take care that the children only receive sustaining and easily assimilable nourishment. The unlimited indulgence which some parents exercise in this respect merely leads to this, that children constantly upset their stomachs with dainties, and generally acquire a repugnance to all wholesome and nutritious food. States of exhaustion and marasmus in a high degree and quite a host of hysterical symptoms sometimes develop in this manner at an early period, which one may rapidly diminish, if it be possible to gain so much authority over a child that it will submit to a rational regulation of its diet. It is no less important in such cases of threatened effeminacy and weakness to attend to the "hardening" of the body against influences of weather, and to their becoming used to muscular exertion. Regular exercise in the open air, cold baths in summer, swimming, gymnastics, and the like, must be very strongly recommended for such children, while allowing them to sit still in the house for any length of time, and anxiously shielding them from every draught and from all exertion in any degree uncomfortible, must be considered decidedly dangerous. Inuring oneself to physical pain in such a case is of itself a procedure tending to corporeal robustness. Particular care must also be taken that the timidity and anxiety so frequently present in nervously disposed children be removed, and that they accustom themselves to suppress such emotions and to overcome their fear of certain animals, of remaining in dark rooms, of being alone, and so forth.

Apparent, however, as is the importance of these principles, it is not superfluous to give warning that the proper limits be not overstepped in such a case, as we so frequently find they are; the hardening process is carried on with a species of fanaticism, which, disregarding the powers of endurance of the individual in question, saps his strength instead of promoting it. It is just these nervous children of whom we are speaking who are so often weakly out of doors; if they are now incited to exertions to which they are not equal, the consequent exhaustion is the greater, and self-confidence disappears, instead of increasing. If they are, notwithstanding, forced by threats and punishment to attempt what they cannot accomplish, one is the more certain to produce natures timid, anxious, and irresolute, and therefore quite under the control of their feelings. People must equally be put upon their guard against accustoming children suddenly and by violent means to frights and surprises. Various cases are known in which just by such attempts (as, for example, suddenly leaving them alone in the dark, or forcing them to touch animals of which they were afraid, and so on) the hysterical predisposition has been confirmed, or the outbreak of severe symptoms actually induced. An influence upon the mental has in the main to be exerted according to the same principles as upon the physical development. Here, too, we must take into consideration aright the individual to be dealt with and avoid extremes. Moreover, it is specially important in this case to eschew the evil consequences of too great endeavors, such as are frequently made by nervous children of precocious intellect, in a measure spontaneously, and partly urged by ambitious parents or teachers.

All these regulations for education are of great importance at the period of puberty, as at this time many injurious influences come into operation which further the development of hysteria. At this period, indeed, it is often too late to combat effectually the seeds of the disease, which have been already firmly implanted. Particular attention must be paid to the chlorosis which most frequently makes its appearance in girls at this age, and which is to be treated on recognized principles. Nothing further has to be added to what has already been said as regards the appropriate physical and mental training at the period of puberty, except that we must avoid allowing children to leave childhood too soon, and that we must carefully strive against the tendency to fancifulness and abandonment to fantastic ideas so frequently met with at this time. Further inferences to be drawn from these principles are self-evident.

Little in particular has to be said of prophylactic measures against hysteria in adult life. These are principally to be taken into consideration when a tendency to hysteria has arisen in consequence of physical diseases or psychical excitement, or even when the disease itself has already broken out, but again disappeared, the tendency to a fresh attack merely remaining. In such case the same rules, on the whole, are to be considered as in the treatment of the disease itself. We must also here consider the advice frequently given, that girls in whom a tendency to hysteria exists ought to marry early, as well as the seeking the welfare of nervous women generally in marriage, that is, in sexual congress. In some cases good may be thus effected, presuming that the marriage is a happy one, and does not in itself carry the elements of such influences as favor the development of hysteria, and presuming, further, that we have to deal with individuals physically strong, full-blooded, and already perfectly developed. Much more frequently, however, is this injurious than beneficial, and it is positively objectionable to advise early marriage in the case of weakly, anæmic girls, in whom the entire physical development is retarded. Even when matters progress favorably at first, we observe almost regularly in such cases that the influences of pregnancy and delivery overtax the existing powers, and cause an outbreak of those symptoms which we had hoped to avoid by the occurrence of these processes.

2. *The Treatment of the Fully Developed Disease.*

This may be undertaken in two ways: first, we may seek to remove the causes which have induced the abnormal condition of the nervous system, and, in the next place, strive to remove this condition itself by means operating immediately upon the nervous system.

a. A removal of the cause of the disease can, of course, only be attempted in those cases in which these are positively known; but even where this is the case, the statement which has been made in considering the prognosis is frequently applicable, viz., that the affection of the nervous system may become independent and persist, although the cause which originated it may have ceased to operate. With this reservation, we must next try to fulfil the causal indications in those cases in which conditions of physical disease are the only or chief causes of the malady. We have most frequently occasion to take the field against states of primary and secondary anæmia, and to employ the dietetic and medicinal agents known as tonic and strengthening. It is for this reason, of course, that the different preparations of iron, and especially the use of ferruginous waters, play so distinguished a part in the treatment of hysteria. In most of these cases it is, moreover, necessary to take into especial consideration the condition of the digestive canal, and the tonic treatment will frequently first be possible after the existing catarrh of the stomach and bowels, the want of appetite, and the hyperæsthesia of the stomachic mucous membrane have been removed by appropriate means. But, as these functional disturbances also arise in consequence of hysteria, it sometimes happens that we can effect nothing against them by the usual remedies, and hence it is indicated that the treatment of the hysteria itself must in the first place be undertaken, that we may then fulfil the causal indication after an amelioration in its individual symptoms. Even in cases of hysteria in which other remedies are indicated, and in which particularly the psychical treatment is of value, the possibility at the same time exists of promoting recovery by instituting a strengthening treatment, calculated to improve the quality of the blood and to increase its entire bulk.

More rarely than for strengthening treatment, there are *indications for diminishing the quantity of blood* in the hysterical, by generally weakening measures or by direct bleeding. The venesections, for the employment of which people had formerly a great predilection, and which were repeated in the case of some hysterical patients with incredible frequency, are at the present day scarcely to be mentioned amongst the remedies employed against the disease. In fact, it is only very exceptionally that indications for it arise, unless the nervous phenomena present may with probability be regarded as the consequence of hyperæmia of the brain, in unmistakably full-blooded individuals, and particularly if in severe convulsive attacks in such people great cyanosis is developed and suffocation threatened. In the extremely rare cases of the latter description, opening the jugular vein in the neck is preferable to venesection at the usual place.—Amenorrhœa, with general plethora, as it is observed in some cases of hysteria (occasionally even as the cause of the plethora), may be an indication for general bloodletting. But in these cases it will be well, in the first place, to try the recognized emmenagogues, savine, etc., and, if these prove ineffectual, to bleed, not by venesection, but by the application of leeches to the vaginal portion of the uterus. We must, however, remember that the failure of menstruation in the hysterical is much more frequently the consequence than the cause of the disease, and that the reddened and bloated countenance which we see in many hysterical people is absolutely no proof of the existence of general plethora, but is much more likely to accompany distinct anæmia. The cases, therefore, in which bloodletting is beneficial are comparatively rare, while, on the other hand, positive injury is very frequently done by this means. This also applies to other debilitating modes of treatment, such as derivatives, the repeated administration of emetics and purgatives, which are still considered by some physicians indispensable in the treatment of hysteria, as also to purulent discharges maintained for a length of time by setons, fontanelles, and the like. Even where these remedies, under the name of revulsives and derivatives, are employed against particular symptoms of hysteria, we see them much more frequently injure than benefit, while they invariably contribute to a deterioration of the general state of nutrition.

The fulfilment of the causal indications is of the greatest importance in many cases in which *affections of the genitals* originate the hysteria. We cannot here consider in detail the different methods of treatment for particular forms of these affections; this belongs to the subject of gynecology. According to the state of the case, the local use of caustics and astringents, of injections and sitz-baths, the introduction of the pessary and sound, as well as appropriate operative measures, will be necessary. The application of leeches to the vaginal portion of the uterus is also much more frequently indicated for inflammatory changes in the womb than for the failure of menstruation with general plethora, as above stated. The examination of the genitals is not to be omitted in any case of hysteria in which there is even a slight suspicion of a diseased condition of these; if such affections exist and are amenable to treatment, this must in the first place be undertaken. In this way, surprising results are not rarely obtained—if not the complete cure of the disease, yet a marked diminution of it, and an amelioration of many troublesome and hitherto very obstinate symptoms. If at the same time the other indications are fulfilled, the general anæmia arising from local disease removed, and proper attention paid to the psychical conduct of the patient, in such cases even a radical cure may be effected.

It is however quite wrong, as does not rarely happen, to use these local measures (injections, caustics, repeated sounding, bloodletting, and so forth), which are only rational in local disease, also in those cases in which the state of the genitals is negative, or at most one of insignificant catarrh, slight change of position of the uterus, without the phenomena of inflammation, or in those due to pressure, and the like. The excessive use of these means under certain circumstances is directly injurious, as they induce diseased conditions, where formerly there were none.

The pains also and hyperæsthesias which make their appearance in the genital and urinary organs without a peripheral cause, as well as the spasms of the same parts, do not by any means always require a local treatment, for, as we have seen, even as symptoms of hysteria itself they may be eccentrically projected phenomena. In such cases, of course, the disappearance of these does not signify a removal of the causes of the disease. It is only rarely possible to fulfil the causal indication in a psychical respect, because we do not as a rule sufficiently know the moral causes of the disease; but even when this is the case, it is not usually in the power of the physician to remove them. Only sometimes can he effectively go to work, if he has had an opportunity of becoming more intimately acquainted with the patient's external circumstances, and of gaining his confidence. The removal of annoying misunderstandings between the patient and his relatives, his liberation from circumstances in which he feels unhappy, his temporary or permanent removal from surroundings injurious to him, the fulfilment of a cherished wish, rendering a longed-for marriage possible, and so on, may under certain circumstances work wonders. But that we may not expect too much in this way, we must remember that the hysterical carry in their own bosoms the seeds of dissatisfaction and misery, and we must not regard as a cause of the disease that which is in reality its consequence.

b. Of the different methods of cure, the object of which is the removal of the disease when it maintains an independent existence, we have in the first place to consider the so-called *specific treatment* of hysteria. The principal remedies which have for ages enjoyed the reputation of specifics are distinguished by a penetrating and in some cases very repugnant odor, while little else is known of their physiological properties which can render their mode of operation intelligible. It almost appears as if more was to be attributed to the stench than to the large quantity of carbon and water contained in some of these remedies, as insisted upon by Valentiner, by means of which they are said to be capable of exercising a peculiar influence upon the fatty constituents of the nervous system. Formerly, when hysterical symptoms were supposed to depend upon the womb wandering about, the opinion was entertained that the latter could be driven out of the positions it had assumed by nasty smells, while conversely, by means of substances having a pleasant odor, in the shape of salves, baths, etc., which quieted the genitals, it could be coaxed to retain or resume its normal position. Asafœtida is perhaps the best known anti-hysterical remedy; it is sometimes to be used in the form of pills, and at other times in that of clysters; it is also one of the ingredients of the aqua anti-hysterica fœtida. Galbanum is used in quite the same way. There are probably few physicians now-a-days who hope by either of these two remedies to be able to perform radical cures; they are most frequently employed as clysters during severe hysterical attacks, but even then are of very problematic value. It is remarkable that some hysterical people do not consider the odor of asafœtida unpleasant. Even at the present day valerian is more frequently used than these remedies. It is sometimes given as an infusion, and again as a simple or ethereal tincture; it is likewise a constituent of aqua anti-hysterica fœtida, and is moreover employed as an addition to clysters, sitz-baths, and poultices. On the presumption that valerianic acid is the active ingredient, various salts of the latter have been recommended for hysteria, especially valerianate of zinc. An anti-spasmodic action especially has been ascribed to valerian; it is therefore considered effectual, particularly in those cases in which general or partial spasms are present; above all, such as occur in the muscular system of the digestive canal. In such conditions, indeed, it often appears to possess a symptomatic value; it cannot, however, effect a radical cure. It is a characteristic circumstance that some hysterical people experience relief from the mere smell of valerian, and that their bespasmed condition may sometimes be ameliorated by letting them inhale some tincture of valerian sprinkled on a cloth. In a like manner, purely symptomatically, but without permanent influence on the disease, do the far-famed castor and musk act in some hysterical cases, both of which, used especially in the form of tinctures, are regarded as specific anti-hysterics. This is also applicable to the other remedies to which an analogous significance has been ascribed, such as the preparations of ammonia, zinc, copper and silver, and also belladonna, stramonium, hyoscyamus, and many others, except that they are almost less effectual even, as regards symptoms, than those previously named.

Particular mention requires to be made of *opium and its preparations*, which have from olden time played a part in the treatment of hysteria, and have also recently been vaunted as a radical remedy for the same. Gendrin maintains that he has cured half his hysterical patients by a continuous use of opium, and Briquet also mentions cures in cases which were not of too severe a nature, and had not already become chronic. It is recommended that one should begin with small and gradually proceed to large doses, and then again slowly leave off. It is correct, as Briquet asserts, that the hysterical sometimes exhibit a great toleration of opium and morphia. If we wish to produce an effect in cases in which sleeplessness, general hyperæsthesia, and the corresponding excitement have attained a high degree, we must rapidly proceed to doses of from three to six grains of opium several times a day, or, with equal frequency, administer morphia in quantities of from three-fourths of a grain to a grain and a half internally or subcutaneously. This tolerance, however, is by no means general. Some hysterical patients may be very easily narcotized by quite small doses; others cannot tolerate the narcotic at all, and suffer from vomiting and other unpleasant consequences which render the employment of this remedy impossible. One must not therefore, in any case, begin with large doses. Now as regards the value of these remedies, they are almost indispensable in the symptomatic treatment of hysteria. In curable cases, as a rule, they greatly assist the plans of treatment otherwise indicated, and in many chronic, incurable cases, they at least afford considerable relief at some stages of the disease, and to some symptoms. But that they can effect actual cures of hysteria, whether by their influence upon the nutrition of the nervous system, or by a permanent diminution of the abnormally exalted sensibility, is, according to my experience, improbable, as such an effect in the case of other mental disorders, in which likewise the persistent treatment by opium and morphia has been extolled as radically efficacious, seems to me unproved. One must, moreover, always clearly bear in mind that by many patients, and especially by the hysterical, the habit of taking opium or morphia once formed can as little be abandoned as that of taking alcohol by chronic drunkards, and that these remedies, after long abuse, cause chronic and incurable states of intoxication, in a manner quite analogous to the former. Although we can, to some extent (by no means always), prevent this abuse in hospitals and asylums for the insane, by discontinuing the use of the remedy, this is much less frequently possible in private practice. It is very dangerous, as unfortunately so often happens, to place the morphia syringe in the patient's own hands.

For the more special indications for the employment of other narcotics and anæsthetics look further on.

The *dietetic* and *hygienic treatment* of hysteria, *in the widest sense of the word*, is in many cases more effectual than the treatment by medicines. As regards this, we have to do partly with producing an effect upon the nervous system by appropriate nourishment, partly with an influence exerted through the sensory nerves, and partly with a psychical influence, without its always being possible to separate these distinctly. Medicinal remedies are of course frequently necessary to assist this. It must in some cases be our first care *to accustom the patient to a wholesome and nutritious diet* (and to do what is occasionally alone sufficient—combat the exaggerated sensitiveness and torpidity). As directions to this effect are most faithfully executed, when patients suppose they are undergoing a special treatment, it is very useful to send them to baths and springs armed with stringent injunctions. Urging them to rise early, to take exercise, and to remain much in the open air is productive of much good. The appetite improves, they are better nourished, sleeplessness disappears, and the confidence of patients in their own strength is restored. If distinct anæmia exists, places where there are ferruginous springs are to be preferred; for the rest it is much less a question of the quality of the waters drank than of the other surroundings. It is for this reason that a suitable residence in the country, especially in the mountains, is often more beneficial than in watering-places, properly so called. It is, in many cases, advisable to send patients to milk- or whey-cure institutions, or to grape-cure resorts. The efficacy of such cures is always greater at these resorts than when used at home. Again, *the systematic external application of cold water* is of material importance. It is beneficial to the majority of those hysterical people in whom a high degree of debility or other diseases do not contraindicate it, if we cause them to wash in cold water daily, and apply friction to the whole body, and during the favorable season of the year to take a cold bath as often as possible. The continuous, and, for particular cases, appropriate treatment in well conducted cold-water establishments is very often of still greater benefit.

People are generally agreed, also, upon the favorable influence of sea-bathing upon the general condition and upon particular symptoms of hysteria. Although there is as yet by no means a perfectly satisfactory explanation of the cause of the efficacy of cold water, yet daily experience teaches us that it can influence in a remarkable manner the whole body, and especially the nervous system. It seems to be a question in part of excitation of the cutaneous nerves by the stimulus of cold, in part of the influence of the latter upon the circulation and the body temperature, and also perhaps of a change in the excitability of the nervous system, produced directly by the cooling. The effects which one usually observes are, diminution of the universal hyperæsthesia and sensitiveness to centripetal excitants generally, increase of the feeling of strength and, at the same time, of the power of will, and therewith the mastery of various other symptoms of disease. Moreover, under the cold-water treatment, systematically pursued, we see, as a rule, the general nutrition improve, which may perhaps depend upon the improvement of appetite which usually takes place, and the consequently greater assimilation of nourishment.

Besides cold water, there are other remedies recommended for hysteria which stimulate the sensory nerves, and whose operation is regarded as derivative or revulsive. The simplest and least injurious of these is the electric current, and especially the induced current. The general faradization recently recommended by Beard and Rockwell, in which the entire cutaneous surface is, bit by bit, treated with tolerably strong induced currents, may sometimes be of use. We also occasionally see the electrical treatment of particular parts of the body, undertaken for particular symptomatic indications, followed by an improvement of the general condition. Yet the uncertainty and inconstancy of this effect seems to argue its occurrence principally in a psychical manner. This applies also to other painful operations: the use of rubefacients to the skin, the application of setons, fontanelles, moxas, and so on, which sometimes, it is true, are of astonishing efficacy, but in other perfectly similar cases utterly fail. Their effect also depends much more upon the moral than upon the physical impression they make.

A proper *moral management* is, without doubt, the most important part of the treatment of the hysterical. Our experience of the most varied remedies is that they owe their efficacy really to the psychical impression which they create; we also see mental excitement of various kinds have so great an influence upon the course and severity of the disease that we may almost accord to such influences a therapeutic value. In fact, although complete cures are rare, yet considerable ameliorations of the disease, and striking results in the case of particular symptoms, may frequently be obtained by this means, and, above all, the operation of other remedies is much assisted by appropriate moral treatment. This itself must vary much, according to the individual case, and only in some respects can it be reduced to general rules.

It is, in the first place, requisite that the physician should either possess the complete confidence of the patient, or that he should succeed in inspiring it. EVERY physician has not "luck" in the treatment of the hysterical, but NO physician has luck in the case of ALL hysterical patients. We meet with such powerful idiosyncrasies in some of these toward particular individuals, that intercourse with the latter becomes impossible, and if the physician is one of these unfortunates it is better for him, as a rule, to give place to another. Sometimes, however, perseverance attains the desired end notwithstanding. But that we may permanently insure the requisite influence, it is absolutely necessary to show the patients that we are convinced of the reality of their sufferings, and do not consider them imaginary as their friends usually do. Nothing is more to be deprecated than unseasonable ridicule. One may frequently lose the confidence of the patient for a long time by an untimely jest. It is, however, equally important to make them understand the nature of their complaint, and to persuade them that they themselves are in a position forcibly to suppress this or that symptom, and also that everything which promotes their power of will also causes an amelioration of the disease. The fulfilment of this last indication, strengthening the will, is, on the whole, the true aim of moral treatment. It can only be accomplished when the physician himself goes to work with this necessary conviction, and is not wanting in zeal and perseverance. He must see to it that his instructions once given are strictly and regularly carried out, and he must gradually make ever greater demands upon the patient's volition. It is, at the same time, his duty to divert the minds of hysterical patients from their sufferings by compelling them to take an interest in other more amusing and useful matters. If we can rouse the ambition and enthusiasm of the patients, it is sometimes possible to make them exert the same zeal in the interest of others which they have hitherto exhibited in bemoaning and exaggerating their own morbid symptoms.

It has sometimes already happened that an "arise and walk" has been successfully said to hysterical patients, who have for years been bed-ridden, and who appeared no longer able to move a limb. It is sometimes the belief in the power of the physician, or in that of other people, or again, in the efficacy of certain remedies that works such "miracles." The greater the assurance with which recovery is predicted, the more certainly does it take place on such a command being given, or in the employment of remedies the most varied. One should, however, refrain from such predictions if he is not sure of his authority over the patient. Without doubt, moreover, the imaginative faculty of the patient is roused to a greater degree by such means as rapidly produce appreciable effects than by those which operate slowly and imperceptibly. Hence it happens that painful remedies, such as electricity, etc., are particularly efficacious, and that patients are much more impressed by the subcutaneous injection of morphia than by its internal administration, and so on. For this reason we must frequently not omit to treat local symptoms of hysteria by local means, although we are convinced of their central origin. When we see, as we occasionally do, a paralysis of the œsophagus cured by simply introducing the sound, or a paralysis of the vocal cords by a single faradization, it would be folly not to make use of these means. But we must remember that local measures redirect the attention of patients to the part apparently affected, and that it therefore becomes our duty in some cases promptly to stop all such treatment, and to urge patients to abstain from the same.

It is sometimes possible by means of threats, or by causing sudden fright, to banish severe hysterical symptoms, and cause the disease to make an important change for the better. Some hysterical attacks are occasionally terminated by unexpectedly soaking the patient with cold water, and their repetition is thereby at the same time prevented. By threatening this procedure, or more powerful skin irritants, or the actual cautery, the epidemic spread in particular of hysterical conditions has sometimes been successfully averted. Amann relates the case of an hysterical patient who suffered from attacks of tetanic convulsions and ecstasy, whom her father cured by a sound thrashing. When we remember, however, that all these potent measures of a physical and moral nature are also active as CAUSES of hysteria, we cannot regard them as perfectly innocent. In fact, the much vaunted showers of water during hysterical attacks do not by any means always give the desired result, but, on the contrary, have frequently been followed by an exacerbation. One can therefore only use such heroic remedies in case of actual necessity when others have failed.

A remark already made must finally be repeated with regard to all remedies operating psychically. They very frequently lose in efficacy by repeated employment, and what has been of use in one attack is not by any means certainly of service in the next. We must therefore always be prepared with a variety of plans of treatment, and not rarely do we see the state of affairs remain unchanged after much trouble and a measure of success.

3. *The Treatment of Particular Symptoms.*

During the hysterical fit special measures are rarely necessary. We must merely take care that the patients do not sustain injury in consequence of their convulsive movements, and that their respiration is not impeded by their clothing. In the very severe convulsive attacks of a more complicated nature, in which patients wheel about and make violent movements in all directions, it is best to let them lie on a mattress spread out on the ground, and to hold their hands just so firmly as to prevent their injuring themselves. Binding them tightly or confining them in a strait-jacket is to be avoided as much as possible, as it may easily prove dangerous by interfering with respiration. Finally, we must not pay too much attention to all these attacks, but make the friends take no more notice of them than is necessary, as otherwise we merely contribute to their exaggeration. If such severe attacks are very frequently repeated we may try to suppress them by suddenly drenching the patient with a bucketful of water; but for reasons before stated we must not make use of this remedy except in case of absolute need. In these, as in milder convulsive attacks, it is quite superfluous to use scented materials and skin irritants, washing the forehead with vinegar, etc., and also clysters with cold water, asafœtida, and the like. Some consider it necessary regularly to shorten the attacks by the inhalation of chloroform, but thereby their recurrence is not prevented and patients are merely accustomed to a remedy which it is difficult again to relinquish.

This is likewise applicable to the use of injections of morphia and clysters of opium. We should only determine to use such remedies in the case of very protracted, severe attacks which are exhausting the strength of the patient. If a peripheral irritation is the cause of the attacks, they may sometimes be suppressed by the removal of such. Thus, in some cases, severe attacks have been averted by the rectification of a misplaced uterus, and so on. The means recommended by Charcot, of exerting powerful pressure in the region of a sensitive ovary, seems unfortunately to be effectual in altogether rare and exceptional cases, and then probably operates in quite the same manner as would any other powerful sensory stimulant.

For the so-called hystero-epileptic fits I have in several cases found the protracted use of bromide of potassium in large doses have the same efficacy as in true epilepsy. As in the latter, the effect seems to be produced in particular cases, and consists generally in a diminution, and less frequently in complete cessation of the fits. After the discontinuance of the remedy, however, these usually reappear in all their former violence and frequency.

In the rare cases in which, from spasm of the glottis during the fit, danger is imminent, the otherwise superfluous inhalation of chloroform and ether is indicated above all else, and, further, the different skin irritants, and, perhaps, also the procedure recommended by Krahmer of passing the finger below the epiglottis and drawing it upwards, may be resorted to.

Simple fainting fits require no particular treatment. On the other hand, in the conditions of syncope with weakness of the heart's action and respiratory movement continuing for days, one cannot omit attempting to produce an effect by powerful counter-irritants, sinapisms, electric stimulation, etc., although these excitants are often perfectly impotent. This applies also to cataleptic fits. If these conditions last for a very long time, it may, moreover, be occasionally necessary to feed the patients artificially, which is best effected by introducing the œsophageal tube, but when the muscles are completely relaxed, it may also be done by passing nutritive fluids through the nose. There will rarely be any occasion in such a case for using nutritive enemata. The constant current applied to the back has sometimes been found efficacious for catalepsy; more frequently, however, it produces no result.

Special remedies are likewise only necessary in some cases for *limited spasms* in particular sections of the muscular system. *Globus* will sooner be removed by an enlightened moral treatment than suppressed by local remedies. Of the latter I have recently seen the frequently repeated application of the constant current effectual in a patient suffering from very obstinate globus (the anode was placed at the side of the larynx), without its being possible for me to maintain positively that the treatment acted otherwise than psychically. For *spasms in the muscular coat of the stomach and bowels*, sometimes warm, at other times cold applications to the abdomen, are found useful; internally, on the other hand, opium and its preparations, belladonna, quinine, valerian, and others, are used with more or less effect.

According to circumstances, clysters of cold water, or with narcotic remedies added, are indicated, or valerian, asafœtida, and castoreum may be used as enemata.

Hysterical vomiting is one of the most obstinate symptoms of hysteria. As a rule, the entire therapeutic armamentarium may be used against it in vain, until it is arrested by some accident. One, of course, always tries in the first place to remove any cause for it by administering food which is as unirritating as possible, milk diet, Liebig's extract of meat, etc., but this is only rarely effectual; likewise ice pills, narcotic remedies of all possible kinds, chloroform, and so on, are usually of quite transitory benefit. In some patients it ceases if we only allow them to eat raw ham, or raw mince meat smartly peppered. In spastic as in paralytic retention of urine, we must not wait too long before using the catheter, for, as Brodie has correctly remarked, we sometimes render retention the more obstinate thereby. In some cases warm sitz-baths, or bathing the entire body, are sufficient to remove this.

Electricity is, apart from the general treatment, the most important remedy in *hysterical paralyses*. Its effect is, however, by no means constant; in two perfectly similar cases, for example, of hemiplegia or paraplegia, it is found in the one to be immediately and completely successful, and in the other to have no effect whatever. After each sitting there is frequently a transitory but well-marked effect, which, however, soon vanishes, and only becomes permanent after numerous sittings. Sometimes, too, it is only possible to remove paralysis in part.

The most effectual plan is, as a rule, the application of strong faradic currents to the palsied nerves and muscles; but, when this method fails, it is sometimes possible to produce more effect by using the constant current. This must likewise be applied directly to the palsied parts with power sufficient to cause contraction, and the circuit must be repeatedly opened and closed. Galvanization of the spinal cord is specially recommended for hysterical paraplegias; this form of palsy is usually much more obstinate than others. Local treatment by electricity is almost always effectual in paralyses in the pharynx and of the tongue, as well as of the vocal cords; but in the case of aphonia especially, there always remains a great tendency to relapses; sometimes, indeed, so great that patients regularly have their natural voice, only for a short time after treatment, and then again become hoarse. External faradization of the neck, with which one sometimes treats first attacks of aphonia successfully, must later be supplanted by intra-laryngeal faradization and galvanization. In all these paralyses, besides the treatment by electricity, passive motion of the palsied parts, and attempts at active movement, are of the highest importance. Powerful compression of the larynx with the fingers sometimes temporarily restores lost speech; in like manner do the involuntary movements operate which take place on introducing the mirror, as also systematic attempts to enunciate. It has been already mentioned that paralytic dysphagia has sometimes been cured by a single introduction of the œsophageal sound. In paralyses of the extremities, also, shampooing and passive movements are of use; the various stimulant and oily frictions probably exercise their influence similarly. Hysterical tympanites may occasionally be rapidly dispersed by powerful faradization of the abdominal muscles, or even by firm compression of the abdomen. In other cases the introduction of the intestinal tube may be necessary. Much discomfort is sometimes caused by obstinate constipation, arising from paralysis of the bowels, which it may only be possible to remove by large clysters and drastic purgatives.

If treatment be at all necessary for *hysterical anæsthesia*, stimulant ointments or powerful faradic currents are used, which in this case can best be conducted to the insensible parts by means of a dry metallic brush.

The *hyperæsthesias* and *pains* of the hysterical most frequently demand the employment of narcotics and anæsthetics. Here, as in other diseases, the use of opium and its preparations for the most varied pains and neuralgias holds the first place. It is used in all possible forms and preparations, being introduced per os or in clysters, as ointments or as a subcutaneous injection.

Many think that these remedies are more effectual for pains, and particularly for neuralgias, if applied directly to the painful parts (that is especially by subcutaneous injection), than if taken internally. The proofs which are adduced in support of this view are not, however, incontestable. But patients themselves believe in the local cure, and therefore it apparently takes place. In reality it is probably always effected through the central organs. Preparations of belladonna, stramonium, hyoscyamus, cannabis, coca, etc., which are likewise frequently employed, are of much less value than opium and morphia. Belladonna is most frequently used in painful affections of the digestive canal. Caffein is supposed to be effectual, especially in painful head affections. The anæsthetic effect upon the cranial nerves ascribed to hydrate of croton chloral, has unfortunately not been established; altogether this remedy does not deserve to be classed as an anodyne. Chloral hydrate, again, has won such a reputation, that in many respects it competes successfully with morphia. This does not, however, apply to its purely anodyne action without simultaneous narcosis. While it is just in this respect that morphia is extremely useful, chloral hydrate can only be employed as a soporific. As such (and combating sleeplessness is often the most important indication in hysteria) it does very good service. Given in doses of one, two, or three grammes, if necessary twice a night, it generally produces a quiet sleep, lasting for several hours, which many patients prefer to the sleep induced by morphia. On the long-continued use of chloral, slight symptoms of vascular paralyses appear, which should be noted; it is, therefore, best to employ the remedy temporarily. Unfortunately, however, habitual drinkers of chloral are already almost as frequently met with as opium and morphia eaters. Inhalations of chloroform are likewise used with transitory success in various painful affections in the hysterical. The remedy is often given internally, especially in hysterical cardialgia. Quinine is sometimes effectual in hysterical as in other forms of neuralgia; after the long-continued use of bromide of potassium, also, we not rarely see both circumscribed pains and states of general hyperæsthesia diminish.

We have already spoken of so-called derivative remedies which seldom remain untried in hysterical pain. They sometimes operate very quickly and completely; more frequently they prove quite inert. The electric treatment also of hysterical neuralgias, with the faradic as well as the constant current, oftener leaves us in the lurch than it cures. Especially have I not been able to convince myself that the galvanic treatment of painful vertebræ, vaunted by Benedikt, is of any particular service. Passive and active motion seems to be especially useful in hysterical joint-neurosis; it is above all things necessary to convince patients of the non-inflammatory nature of their complaint, and to urge them forcibly to overcome their inactive condition.

We cannot here enter more fully into the treatment of the more serious hysterical mental disorders. Patients who are thus afflicted are usually found in lunatic asylums.

DISTURBANCES OF SPEECH.

AN ATTEMPT IN THE PATHOLOGY OF SPEECH.

KUSSMAUL.

CHAPTER I.

Human Speech, the Logos, the Agent by which Definite Thoughts are expressed as contrasted with the Speech of Animals.—Unconsciously evoked, it serves the Ends of Consciousness.

Like human beings, animals, too, possess abundant means of expressing their emotions and desires, and of designedly rendering themselves intelligible, not only to each other, but also to man. Among these may be mentioned touch, cries intended to scare or to entice, sounds of entreaty and of complaint, melodious song, looks, and gestures. To some even organs for the articulation of sound are not denied. But the power of uttering a *word*, which, originating in thought, can in its turn give origin to thoughts and deeds, the *logos* of the Greeks, which includes both *reason and speech*, is only granted to man. Speech is by no means a conscious human invention, as was held by Locke and Adam Smith;[1] it is no work of voluntary combination of positions (thesis), but a work of nature (physis). It originated without consciousness or intention, although it is uttered now with both consciousness and design (Steinthal).

It may, to be sure, on a mere superficial glance, appear an unwarranted conclusion that speech originated unconsciously, inasmuch as conscious and reasoning cognition cannot be imagined without it, and that the whole world of our ideas is constructed by its aid alone. But in this we have nothing but the application of a general principle, according to which all conscious action is appropriately preceded by unconscious. It is not alone our sensory and motor apparatuses that are at first unconsciously and unintentionally trained to certain purposes before they serve conscious ends; the metaphysical forms of cognition also included in the category of cause, object, property, etc., as well as the logical of judgment and inference, find in grammatical forms their expression before an idea of metaphysics or logic exist. Or, as von Hartmann[1] expresses it: "As in the course of time human intelligence began to philosophize it found ready-made a language already richly endowed with forms and ideas."

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[1] *Max Mueller*, Vorlesungen über die Wissenschaft der Sprache. Leipzig, 1863. Bd. 1. S. 26 und Bd. 2. S. 284 et seq.

CHAPTER II.

Speech as an Act of Expression and as a Something Expressed.

Human speech is the result of movements which give expression to thoughts either by indicative gestures, sounds, or written words. Under the term speech we understand then at one moment the *physico-psychical act of expression of thought* by which the latter is converted either into an indicative gesture or an uttered or written word, and the thought-series are worked up into a sentence; at another, *the expressed matter* itself, presented to us as a word according to form, contents, and syntactical arrangement. As a something expressed, then, speech may be regarded as the object of *comparative philology* and *ethnological psychology*, while it is the province of physiology and psychology to deal with it as a physico-psychical act.

If the mental or bodily processes by which speech is produced undergo morbid disturbances, both the form and substance of what is expressed are altered, and the language becomes the study of the pathologist. The latter has then the double task to perform: in the first place to deal with such disturbances in relation to symptomatology, and nextly pathogenetically to refer them back to changes in the organic mechanism of speech which produce them. As elsewhere, so here also we meet with imperceptible transitions from physiological changes to pathological. Physiology and psychology on the one hand, and pathology on the other, serve to throw light mutually upon one another: to them originally we must look for a clear comprehension of the laws governing the formation of language.

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[1] *Edw. von Hartmann*, Philosophie des Unbewussten. Abschnitt B. Cap. VI. "Das Unbewusste in der Entstehung der Sprache."

CHAPTER III.

Speech as a Movement of Expression and an Acquired Reflex.

Those extremely multifarious movements, by which we give expression to the mental processes within us, may be best considered under the heading: *Movements of Expression.* Many of these, as, for instance, the play of features indicating sweetness, acidity, and bitterness,[1] screaming, weeping, and laughing, we have no need to learn; they are *congenital reflexes.* Others, and among them speech, must be learned, and are the outcome of practice. Children learn to speak without being able to account for it, merely owing to unconscious impulse, directed at the same time by external instruction. Within they are impelled to it by feelings and pictorial intuitions which gradually become elevated into intelligent conceptions. The outward instructing agent utilizes this inner impulse (so useful pedagogically from its tendency to mimicry) by every wise means to stamp upon gestures, play of features, and utterance, its own impress. Thus, we may regard speech in its earliest stages as an *acquired reflex.*

The chief characteristic of deliberate intention, which distinguishes between the acquired and the congenital movements of expression, is its greater capability of adapting itself correctly both in form and degree to the purposes which it has in view. Owing to this property, it is rather difficult for us to see in them nothing but a play of mechanical contrivances acquired by practice. And yet pantomimic acts and spoken and written words are nothing but the products of internal self-regulating mechanisms, set in ordered motion by feelings and conceptions, just as one may set agoing a sewing, calculating, writing or speaking machine without needing to know their construction. We can see clearly in this, however, that just here lies the best guarantee for the smooth working of these movements and for the accurate and rapid progress of speech. The will, finding everything already prepared, and having simply to employ the preformed and well-schooled mechanisms to carry out its designs, is able to do so with perfect ease. Just as a leader, in disposing of the hundred thousand members of his well-organized and well-drilled army, has only to issue general and comprehensive orders, so we, for the execution of the most complex series of movements of our organs of speech, require merely to will the expression of a thought by this word or that sentence, in order to actually utter it. Fortunately for us, in so doing we have not to trouble ourselves further about the details in the working of the innumerable telegraphic stations which convey our ideas. If our despatch is properly drawn up and handed in, and the organization is in good order, we may be certain it will reach its address.

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[1] Conf. my "Untersuchungen über das Seelenleben des neugeborenen Menschen. Leipzig, 1859. S. 16.

CHAPTER IV.

Preparatory Stage of Speech in Interjection and Imitative Gesture.—Its Retrogression during Sickness to its Earlier Stage of Development.

In its first, to a certain extent, *preparatory* stage of interjection, imitative gesture or imitative sound, or both combined, speech is a sensation and imitation reflex.

Interjection in its primary form is an *outburst of feeling*—the expression of wonder, joy, or any other emotion originating from the exercise of any of the perceptive faculties. It is consequently no mere sensory reflex, as, for instance, sneezing, in which motion follows immediately upon the tickling sensation in the nose.

And yet there exist transitions from mere sensory reflexes to emotional reflexes—from the cry of pain which both animals and infants will utter, even after destruction of the cerebrum, to the unarticulated cry which escapes the mother when she sees her child in danger. In both cases a painful feeling is betrayed by a reflex cry, but that of the mother is based upon a reasoning cognition in contrast to that of the anencephalous child. One step farther and the reasoning cry acquires the form, in the scale of reflexes, of an articulate complaint, or regularly spoken interjection, as, Alas! or, Good gracious! and such like. The inner motive remains the same, but the motion is no longer the same simple natural utterance, but a word acquired by instruction.

Imitation, again, is not necessarily immediately consequent on sensation; it always implies a certain amount of attention and perception and *pictorial intuition* gained thereby. But while interjection is at first purely subjective, and but expresses some feeling of the individual, imitation contains an essentially *objective* element, endeavoring as it does to reproduce in kind a given perceived phenomenon by gestures or sounds.

Interjections and imitative gestures and sounds are the earliest germs of pantomimic and phonetic speech, but as yet not fully these. Speaking involves *understanding* oneself as well as others. We speak for the first time when we comprehend our own feelings and intentions, and are able to exchange with others true, intelligent conceptions, no matter whether this is effected by gestures or sounds. Interjection and imitation constitute the bridge leading up to this *comprehension*. Interjection and onomatopoësis supply to the thinking intellect, which transforms feelings and intuitions into intelligent conceptions, the first words, or, more strictly speaking, word-germs.

Speech is only possible where the individual lives, feels, imitates, and thinks, *in the companionship of his fellow-men.* Strictly speaking, it is not a product of individual man, but of *peoples*. From the fact that in the earliest ages all men occupied an equally low stage of development, there was of course, among the similarly living, feeling, and perceiving members of the community a mutual understanding of each other's outbursts of feeling and imitative gestures and sounds; and so these came to correspond in their most essential characters. Thus, not only did sneezing and screaming come to be understood by every one, but also such gestures and emotional sounds as those of enjoyment, horror, etc., or the mimicry of cock-crowing, or barking of dogs. Even at the present day savage peoples render themselves easily intelligible to one another by means of gestures and imitative sounds.[1]

Some distinguished philologists of our day, and among them Max Mueller and Lazarus Geiger,[2] have improperly denied this origin of language from interjection and imitation, or, at all events, have estimated its significance at almost nil. To be sure, words of the present day display, as a rule, nothing of the original relation which must have existed between the perceived phenomenon and the feelings aroused by it, or the phonetic movements called forth by it from the first word-maker. In the course of ages they have assumed a marvellous diversity of form in the mouths of the different nations. But, for all that, a large proportion of words in all the languages of the world have still preserved a certain sound which at once betrays their sense.[3] In our children, however, the *onomatopoetic* feeling persists still in undiminished force, and their conception of a dog, in spite of all Mueller's ridicule of the "bow-wow theory," proceeds invariably in the first instance from sound-mimicry. It is also certain that speech is learned only by onomatopoësis: the child mimics its parents' words just as it does the barking of a dog or bleating of a sheep. The same feeling makes itself strikingly evident also in the constant transformation of words and the reception of strange ones into the peculiar glossaries of individual peoples.[1] Finally, the art of speaking, whether in poetry or prose, appeals again and again to this strong feeling.[2]

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[1] Even deaf-mutes can render themselves easily intelligible to savages. The pantomimic expressions of savages and deaf-mutes are analogous to the dialects of a primal language. Conf. some interesting facts related by *E. Tylor*, Investigations of the Early History of the Human Race.

[2] *L. Geiger*, Ursprung und Entwickelung der mensch. Sprache und Vernunft. 2 Vol. Stuttgart, 1868; also by the same author, Der Ursprung der Sprache. Stuttgart, 1869.

[3] Exemp. g., our "pah," "phew," "tic-tac," "clip-clap," "swish," "hum," "crackle," "rumble," "cluck," "pewit," "cuckoo," and so on.

Again, *writing* springs out of that impulse which urges us to reproduce all phenomena in mimic representations. But even so far back it was a more lofty and artistic impulse which directed the hands of the ancient inhabitants of the caves, when they executed the portraits of their contemporaries, the reindeer and the mammoth; for here there was no rude emotional impulse, no question of satisfying any necessity of daily life by the production of such drawings. It is no doubt a long step from the pictorial representation of a perceived object to that of a heard word or phonetic element; for written language involves the analysis of the structure of the language, so far as words, syllables, and accents are concerned, which only becomes possible at a stage of culture attained later on. And yet the earliest germs of written language are found in this artistic impulse of the first inhabitants of the earth, whilst its perfecting was reserved for a period of much higher intellectual development. The hieroglyph is the parent of phonetic word-writing. Here then we have an early bond between gestures, words, and writing; from one soil they have sprung, three sources of intellectual power—the youngest, writing, having come forth in the full sunlight of completed consciousness.

In conditions of either transitory or permanent mental weakness, speech may often be observed to *revert again to the level of its preparatory stage.*

Many insane people, for instance, often give vent to their emotions in interjectional expressions of feeling, at one time in the form of simple sounds, especially vowels; at another, of syllables or word-like combinations of sounds. These are often entirely unassociated with ideas, or only so with obscure conceptions.[1]

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[1] For instance, out of the root tan (to make tense) is formed the (German) word Donner (thunder); and the French rouler (from rotulare, rota=a wheel) is metamorphosed into rollen, instead of into ruhlen. Conf. *Oscar Peschel*, Völkerkunde. Leipzig. 2. Aufl. Entwickelungsgeschichte der menschlichen Sprache. S. 103-117.

[2] In the words of a great German poet, "Die Werke klappern Tag und Nacht," *Steinthal* remarks, that not only the word "klappern," clatters ("klappert"), but also the words "Werke" and "Tag."

Attention to those imitative utterances to which the name of "*echo speech*" has been given was first drawn by Romberg.[2] He says: "Sick people sometimes repeat monotonously words and sentences uttered by any one close to them, without giving any evidence of their attention being aroused, and without connecting any ideas with what they say." He adduces several instances of this, and Bateman[3] has lately placed on record a new and most striking case of it which occurred in Voisin's wards in the Salpêtrière. Many similar observations by other writers might also be mentioned.

CHAPTER V.

The Principle of Sound Metaphor.

Although not difficult to conceive why man should respond to the reception of sounds through the auditory apparatus by making imitative noises, it is so to understand why sounds should be chosen as the favorite interpreters of *all* the senses; yet there is no people on the face of the earth, however savage it may have remained, which contents itself with indicative gestures. In every quarter of the globe man has acquired a readiness, through the agency of the voice, with its extraordinary variety of sounds and combination of sounds, in ascertaining the condition of all the senses, and of copying down, as it were, their pictorial intuitions. Whence springs this natural pre-eminence of the *voice* over gestures as a means of intellectual intercourse?

The sensations through which we obtain a knowledge of things outside ourselves, however various the forms they may assume, according to the nature of the senses affected and of the stimulus arousing them, may awaken, nevertheless, in the perceiving individual very much allied feelings of pleasure or the reverse, even to the extent of pain. We know, for instance, that a startling color may arouse the same feeling within us as a startling sound, and the one sensation reminds of the other, although conveyed to us by a different sense and determined by other impressions. We institute comparisons between sensations according to their relationship in feeling, and so speak of crying colors and cold lights, bright, warm, high, and low tones, sweet sounds, hard and soft noises, sharp odors, mild taste; and, as these examples show, not only do the simple sensations become intuitions gained from the senses by tentative movements, but they are helped by unconscious judgment as well. These intuitions, then, convey the general impression of simple sensations, such as roughness, hardness, depth, etc., when investigated in respect to their relationship of feeling, and are, according to the result, considered similar or dissimilar. To put the matter figuratively, we are able to translate the feelings of one sense into the speech of another.

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[1] *Martini*, Veränderungen der Ausdrucksweise bei Irren. Allgem. Zeitschr. f. Psychiatrie. 1856. Bd. 13. H. 4. S. 605.

[2] Lehrbuch der Nervenkrankheiten. 3 Aufl. S. 655.

[3] On Aphasia. London, 1868. p. 75. Conf. *Brosius*, Ueber die Sprache der Irren. Allgem. Zeitschr. f. Psychiatrie. 1857. Bd. 14. H. 4. S. 63.

Manifestly, of all the senses, that of hearing presides over the richest range of feeling; of all the arts, music, which by its harmonies can stir up the ocean of feeling to the greatest depth. But the power of mightily influencing an emotional condition is not restricted to musical sound alone; noises also possess it to a very large extent, and may excite the phantasy powerfully. Of all hallucinations, those of hearing are most feared by the mental physician. It is in this extraordinary richness of the hearing sense in shades of feeling, then, that its capacity lies of participating to a certain extent in the impressions of all the senses, and hence the voice, although primarily but the interpreter of the feelings aroused through hearing, is, nevertheless, peculiarly adapted to express also the feelings of all the other senses. Furthermore, spoken words convey the impressions of all the senses with the least expenditure of force and time.

This is what was understood by Heyse[1] as the *principle of*

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[1] System der Sprachwissenschaft. S. 94.

sound metaphor: "A perception of any one sense is expressed by a phonetic composition which produces upon the inner sense of the hearing apparatus a similar impression to that which the perception to be indicated evoked through that other sense."[1]

Kleinpaul[2] regards it as "almost an accident that phonetic speech should have come so exclusively into use, since there is very little doubt that, had pantomimic speech been developed by the use of centuries, it would hardly have been inferior in completeness, adaptiveness, and variety." But, in spite of the interesting support which Kleinpaul gives to the theory of the great capability of development inherent in pantomime, he has not convinced us of the identity in value between the speech of gestures and that of phonation. Deaf mutes, he maintains, may produce a piece of Shakespeare in the language of signs; but, though this may be true there, proof is still lacking that pantomimic language will ever raise up a Shakespeare among the deaf-mutes.

CHAPTER VI.

Origin of Intelligent Speech from Word-roots.—Intuition and Intelligent Conception.—Sensory or Instinctive in contrast to Intelligent Judgment.—Speech as an Associated Conception-reflex and as an Act of Will.—The Three Stages of Language: Preparation, Diction, and Articulation.

The speech in which we now express our feelings and thoughts has long forfeited its originality. It has come down to us as an inheritance which, having passed through the hands of thousands of generations, has undergone innumerable modifications. To discover the internal and external determining causes of these latter is the province of philology, and concerns us no further. The onomatopoësis of the infancy of the human race has, with the exception of some faint traces, vanished from the languages of the nations. Even our interjections have little any longer in common with the utterances of feeling of those earliest days of mankind, and they have only partially preserved their naïve character. Do we not see this in the oaths of reprobates swearing by the Holy Sacrament and profaning the holiest names in Christendom? Dogma and church history supply them here with suggestions for interjectional relief in moments of passionate excitement.

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[1] I have written to Prof. Kussmaul to know if this quotation has been correctly translated, or whether there was a misprint in the German text, as I felt it to be extremely obscure. He replies, thanking me for pains I had taken with the quotation, which, he says, he has put in inverted commas, to show that "they are Heyse's own words," as he feels "that they are not so clear as they should be, and that a great philologist like Heyse ought to have expressed his thoughts with more clearness and accuracy." He says this translation "appears to express what Heyse meant to say."—TRANSLATOR.

[2] On the theory of pantomimic speech, conf. Zeitsch. f. Völkerpsychologie. Bd. VI, 1869. S. 353.

So far as comparative philology has succeeded in following up to their earliest etymological roots the languages of different nations by means of a comparison of the oldest literary remains, we stumble everywhere upon *well-established nuclei of language* which still display, though but partially, their original onomatopoëtic character. Even these *word-roots* in a philological sense are far more than mere sounds expressive of feeling or imitation; they are "*thought-nuclei*" around which, as developmental centres, the whole world of ideas whose possession is the joy of humanity at the present day gradually sprang up in word-structures.[1] They are the *idea-elements of speech*, as consonants and vowels are its sound-elements. In them may be seen faithfully reflected the condition of intellectual development of the progenitors of our great national families (Indo-Germanic, Semitic, Turanic, Chinese, etc.); and etymology in following up the history of the changes in such ancient thought-nuclei, through all the phases of the life of these peoples down to the present day, becomes the history of the genesis of ideas as completed in the families and later peoples (Steinthal).

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[1] The Aryan root MAR signifies to grind, to pulverize. From it are derived the Sanskrit *malana*, to grind or rub; and a number of words all signifying *mill*, the Latin *mola*, the Greek *myle*, the Irish *meile*, the Bohemian *myln*, and the Lithuanian *malunas*. Then the German *Müller* (miller); *Mühlstein* (mill-stone); *Mehl* (meal); mahlen (to grind); Mahlzähne (molar teeth, dentes molares), etc. Metaphorically, MAR acquired the signification of being ground, pulverized, worn out, destroyed. Hence the Greek *maraino*, to wear out; *marasmus*, wasting; *morior*, I die; *mors*, death; *morbus*, disease. In Sanskrit, *mriye*, I die; *márta*, man considered as a mortal. In old Slavic, *mřeti*, to die; *moru*, pestilence, death. In Lithuanian, *mir-ti*, to die; etc. In these examples one sees the aptness of Jean Paul's comparison of language to a lexicon of obsolete metaphors.

With the creation of word-roots as established speech- and thought-nuclei, language was elevated from the preparatory imitative stage to the second *real word-forming* or *intelligent stage*. Arrived here, speech is no longer the mirror of mere sensory intuitions. The individual has now at his disposal intelligent conceptions which find in words a palpable form cognizable to the senses. An *intuition* is not an *intelligent conception*. The former is nothing but the sensory picture, the photograph of things as they happen to present themselves accidentally, or were viewed and copied as a certain combination of dots and lines of light and shade, of white and black and various colors optically, or a definite combination of tones of different degrees of height, loudness, of body and timbre acoustically. But it is not yet the true picture of the object such as we throw off by our own creative power, marking all its essential characteristics and rejecting all which appears to us accidental and transient about it, and this after comparison of numerous tentative intuitions under different circumstances and conditions. Let a photograph be never so successful technically, it fails to satisfy us unless it so represent the object that we at once recognize it in its most essential features. No one would gratify a friend with a portrait depicting him in the act of making a grimace, even though technically it might be a masterpiece of photography.

But we must admit that even in the creation of pictorial intuitions, judgment is already more or less mixed up. The actual shape of the object, the resistance it offers to the touch and to attempts at lifting, the intensity with which and the direction from which the sound reaches us, we unconsciously estimate with great certainty. Animals deprived of their cerebrum and unable any longer to execute voluntary movements still possess visual intuition, which supplies them with the power of suiting their reflex movements to surrounding circumstances (Goltz). There exists, then—a lower, let us call it—a purely sensory or instinctive analysis and judgment, discrimination and combination, which has within the brain an origin widely separate from the higher and intelligent. Such a *sensory* or *instinctive analysis* and *judgment* operates, no doubt, in those reflex sounds which adapt themselves imitatively to certain perceptions; but the formation of words which have their origin in conception is brought about by *intelligent analysis, comparison,* and *judgment.* Words are more for us than mere pictures converted into sounds of things mirrored on the senses; they are *pictorial symbols* for things as we have come to conceive them through deduction based upon the incessant variation of their phenomena—they are the *symbols of ideas* (Begriffszeichen).

Based upon the world of sensory intuitions, which was the first to open upon him in a full blaze of color, man builds another colorless world of abstract ideas, but not in such a manner as to demolish the first formed by the last; for the sensory pictures, though gradually paling, live behind the ideas, and renewed perception freshens them up easily to their original vividness. If the picture grow indistinct, the sharpness of the idea-outlines obtained from it suffers in consequence, for the world of ideas has the roots of its power in the world of sensory pictures. Consequently it is correct to say that the senses supply us with all the matter recognized by us, but the intelligence assimilates it according to the *formulæ of cognition* with which it is supplied. The material acquired by the senses must first pass through the three-fold mill of logic, metaphysics, and grammar before it really becomes the property of the intelligence.

From all this it is clear that words, so soon as speech has passed the preparatory stage of onomatopoësis, are no longer mere intuition reflexes. They rather represent the abstract conception or idea upon which the words are dependent; and between the sensory picture and the word there exists a wide field of intellectual work which must always be engaged before a word can be uttered. *The latter is always the expression and conclusion of a movement of thought, which has, no doubt, its primary source in sensory perceptions, but is not necessarily immediately dependent on these.* Thoughts generate thoughts as surely as thoughts are generated by perceptions through the senses. For this reason, since a distinction has been made between intuition and conception, words have been very properly regarded as *conception reflexes* (Steinthal, Lazarus).

When we employ this expression we only mean that the *reflex mechanism*, which executes all the organic movements emanating from the nervous system, also generates the complex movements of the words which express the intelligent conceptions. *To reveal this reflex mechanism in the whole motion of its wheels and transmissions, and the vital forces by which it is fed, is the province of the physiology of speech.* In speaking, we see conceptions, feelings, and sensory intuitions interlacing with one another in a most extraordinarily intricate manner, and so setting agoing the motor apparatus which accomplishes the audible utterance of the word. We only apparently simplify the matter by characterizing speech straight off as *an act of the will*, or by classifying it according to physiological terminology under the *associated conception reflexes*. What is called *will* is no simple force, but a very complex dovetailed combination of partly *associative*, partly *reflex* processes in the excited nervous system; and speech as an act of will is only then explained when we are able to localize in the nervous system all the operative special associations and reflexes—that is to say, to determine anatomically all the paths by which the stimulus is conducted, and at the same time to establish physiologically all the sources by which it is fed.

It is already a long established psychological axiom, quite apart from all nerve physics, that a bare idea, although it may qualify the will for the highest efforts, never alone determines a man to act, but that it is always some feeling that does so, such as of duty, of justice, of pity, etc. A conception emanating from sensory intuition as a cold and colorless abstraction, has as such no impellent motor power. Again, we see when an idea is expressed in speech that the prime mover of the uttered word is invariably a feeling. But even this is not sufficient. For speaking are further requisite symbols, intuitions of the intelligent conceptions themselves which we have put into the sensory form of word-pictures. Only thus do we apply the reflex lever to the motor apparatuses of the organs of speech. But this psychological comprehension of the subject is still no physiological one. For what we characterize as feeling, conceptions, and intuitions, are to the physiologist but the psychical expression of material processes in the organic groundwork of the nervous system, which is equally adapted for the evolution of the forces belonging to mechanical and psychical performances. These mechanical and psychical performances course inseparably side by side, united by the same set of laws; and to understand both, it is first necessary to lay bare the organic conditions in the nervous system out of which both together spring twin-like. It is the task of the physiology of speech to ascertain where and how the nerve-substance is rendered capable by conceptions and feelings of creating language through the medium of word-pictures.

Let us now reconsider for a moment the most essential points in the process of talking. In the first place, a *thought* is indispensable which we have *conceived*, and then an *impulse of feeling* urging us to express it. Next we *choose* and *say* the *words* which the acquired language in our memory places at our disposal. Finally, the *reflex apparatuses* are called into play which give outward utterance to the words. Accordingly, the act of speaking consists in three stages or processes: (1) The *preparation* in the intelligence and feelings *of the matter to be uttered;* (2) the *diction* or the formation of the words internally, together with their *syntax;* (3) the *articulation* or formation of words outwardly, irrespective of their connection with one another in the matter spoken.[1]

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[1] These three definitions do not appear to the translator to be as carefully expressed in the original as might be, but he has thought it his safest course to translate them as literally as possible. The word diction is the same word as that used in the original. —TRANSLATOR.

CHAPTER VII.

Speech as the Agent of Intelligent Cognition.—Dialectical Thought as opposed to Thinking in Objective Images.—The Relative Independence of Conceptions upon Words.

In characterizing it an associative reflex we have not exhausted the meaning of speech. It is more than merely a motor process reflectively bound up with thought by associated sensory symbols. It is besides this a mental act, without which an intelligent cognition of things cannot be attained. It not only reproduces what has been thought, but claims thought and generates thought anew; and *comprehension of what is thought* only becomes possible through speech.

Animals are capable of forming conceptions, judgments, and conclusions of a simple kind, human beings alone of ideas and more highly compound judgments and conclusions. The dog bounding to the door, with a joyous wag of his tail, as he sees his master put on his hat, has a conception of what is taking place (Wundt[1]). Pictures arise in his recollection, and he judges correctly that his master is preparing to go out; to this he adds the other judgment, namely, the expectation that he will be allowed to accompany him. But these conceptions of animals are but little elevated above the intuition world of the senses. Their thought only deals with objective images and very simple abstractions, that of man with word-pictures and ideas. Combining his conceptions with words, he creates not alone short symbols intelligible to others, but imparts to his abstractions the completeness, accuracy, and stability of ideas. Only as an idea is the conception capable of a critical analysis and logical dissection. It is this which distinguishes man from the brute creation, namely, that he is not only able to form ideas and judgments concerning things around him, but is also able voluntarily to array these ideas and judgments as objects before himself and others in a form cognizable to the senses, and to test their accuracy dialectically.

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[1] Vorlesungen über die Menschen- und Thier-seele. 2 Bde. Leipzig. 1863.

It is still a vexed question *whether intelligent thought is bound up with words* (Condillac, Max Mueller, Bastian, and others), *or is quite independent of words* (Locke, Helmholtz, Maudsley, Finkelnburg, and others).

But it is clear that conceptions and words are different things, and the *independence of conceptions upon words* may also be easily proved. For instance, the Lord's Prayer expresses in the most diverse forms and combinations of words in different languages the same religious contents. Newly discovered animals and invented machines are earlier recognized than are their names (Tylor). We read aloud to another, thinking all the while of something else, and afterwards do not know what we have been reading about (Finkelnburg). But the most instructive fact is one which each may verify on himself. We can, for instance, recall vividly to our minds all the essential characteristics of some person or thing, can accurately conceive a picture of it, or perhaps set down such on paper; we know how it affects us, but we have forgotten the word descriptive of it: this is an "aphasia," which falls quite within the range of physiological disturbances. Conversely, we may not be able to recollect any longer the meaning of a foreign word or artistic expression; or again, onomatopoëtic and etymological feelings lead us astray to false and often ludicrous constructions. At times, too, thought travels so fast that speech is unable to follow it up.

But not alone is the independence of conceptions upon words beyond doubt, but, as has been adduced from the facts put forward by Tylor—and the development of conceptions in children and animals likewise proves it—*conceptions may originate without words.* One thing is certain, however, namely, that the conceptions of animals remain far behind those of human beings, and that only in the latter do they attain to entire clearness, delicacy, and well-rounded form as *ideas* through the medium of speech.

By way of proof that intelligent thought is not bound up with speaking, and is quite independent of words, a number of facts are quoted, to which we must now devote our attention more closely.

1. It has been pointed out that from birth on, *deaf-mutes* have learned to recognize things and relations properly. A case related by Kruse,[1] bearing upon this point, is particularly instructive.

A deaf and dumb boy was found by the police, in 1805, straying about Prague. Not being able to make anything out of him, they placed him in an institution for deaf-mutes, where he received instruction. When sufficiently educated to give accurate answers to questions put to him, he gave a description of as much of his former life as remained in his memory. His father, he said, owned a mill; he described the furniture of the house and also its surroundings minutely; he gave a full account of his life while at home: how that his mother and sister died, and his father married again; how that his step-mother ill-treated him until he ran away. But he neither knew his own name nor that of the mill; he knew, however, that it lay to the east of Prague. Inquiries were made, and the statements of the boy were found to be correct. The police found his home, gave him his proper name, and secured to him the succession to his father's property.

From this we see that a deaf boy who had not learned phonetic speech had nevertheless stored up a multitude of accurate recollections, and was able by the exercise of judgment to arrive through them at correct conceptions of very intricate relations. But whether he found these conceptions without the slightest aid from the language of gestures is not related. There is no deaf-mute who has not learned a number of gestures by means of which he is able to make himself intelligible, if not to strangers, at least to those belonging to him, unless, indeed, he be idiotic too. The objection is also admitted that this boy, after being instructed in the institution, may have subsequently brought many recollections into their proper connection, and now for the first time have understood formerly unintelligible relations. We are justified in this conclusion by the combined testimony of such experienced instructors of deaf-mutes as the Abbé Sicard, and Eschke, the director of the Berlin establishment for the deaf and dumb, that deaf-mutes without instruction hardly rise above the level of animals, as regards reason and feeling.[2]

Another case is also put forward, namely, that of Laura

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[1] Ueber die Taubstummen u. s. w. Schleswig. 1853. S. 54.

[2] Conf. *E. Schmalz*, Kurze Geschichte und Statistik der Taubstummen-Anstalten u. s. w. Dresden. 1830. S. 26. f.

Bridgeman,[1] who became *blind and deaf* in her second year, the only other sense remaining perfect being that of touch, those of smell and taste being, the former almost, the latter to a very large extent, destroyed. This girl attained nevertheless to a high grade of culture, was able to grasp the most abstract ideas, *e.g.*, of God and of immortality, and eventually became a schoolmistress. But this example is one of the very best proofs that speech is indispensable for intelligent cognition.

This remarkably intelligent person, although incapable of acquiring phonetic speech, became possessed, thanks to the care and energy of her teacher, Dr. Howe, of the whole world of thought around her through the medium of the language of touch. In spite of her high mental gifts, of a remarkable memory, a most lively power of imitation, and a marvellously accurate sense of touch capable of almost any amount of development, she hardly occupied a higher place in her home than that of an intelligent animal upon whose instruction one has bestowed much labor, and this, although she was the object of the tenderest maternal solicitude. She distinguished the objects about her home according to their form, density, weight, and heat, and imitated the movements of her mother by an examination of her hands and arms; she even learned to sew and knit a little. But in a few months her psychical condition underwent a marvellous change at the hands of Dr. Howe, to whom she came when seven years old, and this through the agency of speech. He affixed to a number of common objects, such as knives, forks, spoons, keys, and so on, labels on which the name of the article was written in raised letters. Laura soon remarked that the crooked lines of the word spoon were as different from the crooked lines of the word key as the shape of the spoon was from that of the key. After this some small labels with the words printed on them were put into her hands. She soon noticed the correspondence of the written words of the label with those on the various objects mentioned, and gave proof of this by placing the label key upon the key, and the label spoon upon that article. All this time she was encouraged by marks of approbation, such as patting on the head, etc. This method was gradually extended to all the objects she could take in her hand. Later on the separate letters were placed in her hand and there arranged into such words as book, key, etc.; they were then thrown into a heap together, and she was left to put together the words book, key, etc., for herself.

"Up to this," says Dr. Howe, "the proceeding was only a mechanical one, and the result was about as great as if one had taught a number of tricks to a clever dog.

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[1] Her history has been related in several reports by her teacher, Dr. Howe, President of the Institution for the Blind, in Boston. Conf. Zeitschr. f. d. ges. Medicin von *Fricke* und *Oppenheim*. Bd. 13. 1840. S. 1.—*Froriep's* neue Notizen. Bd. 21. 1842. S. 273.—*Boz*, America, translated by Moriarty. Th. 1. 1843. S. 56.—*Burdach*, Blicke ins Leben. Leipzig. 1844. Bd. 3.

> The poor child had sat there in mute astonishment, and patiently imitated everything that was performed before her. But now the matter seemed to dawn upon her in its true light, her understanding began to exercise itself, *she noticed that she now possessed the means of arranging for herself symbols of something that lay before her mind and of showing this to another mind; immediately her countenance beamed with human reason;* she could no longer be compared to a parrot or a dog; the immortal intellect now seized greedily upon this new bond of union with other intellects! I could almost point out the moment at which this truth dawned upon her and poured light over her whole face."

No more beautiful example could be given than this remarkable physiological experiment of Dr. Howe, of the *cultivation of reason, the formation of the world of ideas, through the agency of words.* We can now see why the Greeks comprehended in the term "logos" reason as well as simply a word.

3. Another instance may also be cited in the case of those *children who, although endowed with hearing, are unable to learn to speak at all or very imperfectly; they appear quite intelligent, understand what is said around them, obey orders, carry out instructions, and so on.*[1] This, however, has all been learned in intercourse with intelligent, speaking persons, by the aid of words rendered clearer through gestures, which the children, too, had come to understand. The words remained in the memory united with intuitions, and associated themselves with intelligent conceptions, or generated such. Thus, even the dog, as a companion of man, becomes wiser and learns to grasp the meaning of many words. But, of course, the more highly endowed human being must profit much more by such instruction than the unreasoning animal, even though, as a consequence of faulty organization of the internal apparatus of speech, he may never be capable of representing by movements the word-pictures he has laid hold of; unless, of course, the intelligence, too, is early wrecked.

4. Finally, the case of M. Lordat,[2] Professor of Medicine at Montpellier, is cited, who, after a fever, suddenly lost all power

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[1] Conf. *Waldenburg*, klin. Wochenschr. 1873. No. 8; also *Broadbent*, Med. Chir. Transact. Vol. 55, p. 146, Case 4.

[2] *Lordat*, Analyse de parole, etc., pour servir à l'histoire de l'alalie et de la paralalie. Montpellier. 1843. More at length by *Proust*, Archiv. gén. 1872. p. 666, et seq.

of speech for several months. In this condition he was so completely robbed of his memory for words that he could not even understand those spoken to him. He is stated, however, to have been able carefully to consider his own condition, to combine his thoughts properly, and even to follow out the thread of his lectures as well as before the attack.

Trousseau[1] remarks, however, that Lordat may have deceived himself as to the acuteness of his thinking powers during the attack, for during the rest of his life there remained behind a certain weakness of his mental power.[2] For instance, Lordat, who had hitherto shone as an extempore speaker, was henceforth unable to improvise his lectures, and had always to read them.[3]

Moreover, before the attack Lordat was in possession of *a world of thoughts acquired by means of speech.* No one will deny the relative independence of conception upon speech in its most abstract intelligent form. It may be the case, then, that, in spite of the total destruction of the memory for words, there may have been still preserved in the mind of such a highly developed thinker abundant accumulated material of thought. We will even admit that the thoughts moved along in the old groove; but we cannot believe that this took place with the same certainty and smoothness as of old, or that Lordat was capable of creating new ideas.

Again, this personal observation of Lordat stands alone, and, as he was a decided spiritualist and opponent of the organicism of the Parisian school, his can hardly be regarded as entirely unbiased evidence. Other thinkers and physicians, such as the celebrated Spalding, of Berlin (1772),[1] and the Parisian professor of medicine mentioned by Trousseau,[2] who had both lost their memory for words in transient attacks, without forfeiting the power of observing and of judging themselves, and of communicating their wishes to those about them by means of signs, did show a diminution of their intellectual acuteness and a certain confusion of thought.

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[1] Clinique méd. T. II. Art. LVIII. "Aphasie." Bull. de l'acad. de méd. T. XXX. 1864–65. p. 672.

[2] Lordat relates: "En réfléchissant sur la formule chrétienne, qu'on nomme la doxologie, 'gloire au Père, Fils, et Saint-Esprit, etc.,' je sentais que j'en connaissais toutes les idées; quoique ma mémoire ne m'en suggérât pas un mot. . . ." On this, Trousseau remarks: "J'avoue ne pas comprendre qu'on puisse songer à une formule de langage sans se rappeler aucun des mots qui la composent." For myself, I have the same feeling as Trousseau. I cannot understand how one can conceive a *formula without symbols*, a *word-formula without words.*

[3] It is also remarkable that Lordat should relate of himself: "L'air stupide, etc., . . . faisaient croire à plusieurs qu'il existait en moi un affaiblissement des facultés intellectuelles." Aphasia, without an appreciable disturbance of the intelligence, does not usually so completely obliterate the latter from the expressions of the countenance.

In our opinion, there is nothing to be learned from Lordat's record of his own case (even suppose we accept it in good faith as correct in all respects) but this, *that ideas, when once acquired, possess a certain independence of words, but not that they are acquired without the help of words.* Objective images and simple conceptions, such as may be aroused even in an animal, are called into being without words, through the medium of sensory impressions; and yet any one may daily observe for himself, among his domestic animals, that here, too, indicative signs, such as pointing to the door or the food-pan, are an essential item in the generating of a conception. But ideas are only acquired through words of sound, of gesture, or of writing.

It is quite true that persons deprived of the power of speech by disease need not have lost their intelligence in the same measure. They not unfrequently still display a certain power of insight into things around them by the expression of their face, by gestures and acts. Numerous observations by trustworthy physicians may be cited in support of this, as, for instance, the following by Broadbent:

A woman of seventy had lost, in consequence of apoplexy, accompanied by transient right hemiplegia, the power both of speech and of writing, permanently. She was totally unable to write, while some words, produced in the form of rapidly uttered interjections, still gave expression to her feelings (*e. g.*, oh, shameful, shameful! nasty, nasty! pity, pity! that's right! etc.). She could read, and displayed great energy and intelligence in trying to regain her right to administer her own property, which had been denied her, on the wrong supposition of her weakness of intellect. Three years later she succumbed to apoplexy, with left hemiplegia. Two apoplectic cysts were then found in the upper border of the fissure of Sylvius, on the left side, one in the posterior part of the third frontal convolution, one further back, including some of the convolutions of the island of Reil, and the corpus striatum was atrophied. The cause for the last apoplectic seizure was not discovered.

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[1] Spalding imparted his experiences on the afternoon of the same day to his friend Sulzer. A notice of these may be found in *Moritz*, Magazin für Erfahrungsseelenkunde. Bd. I. St 2. S. 38.

[2] Rostan was the professor! Conf. also the statements of a physician who occasionally suffered from such aphasia, in *Westphal*, Verhandlungen der Berliner Gesellschaft für Anthropologie. 1874. S. 101; and those of another educated man, in *Spamer*, Archiv für Psychiatrie. Bd. VI. S. 531 and 540.

Such cases force us to the conclusion that, although we arrive at our ideas through speech, these, as soon as they are formed, possess an independence of words. *Sensory pictures, feelings, movements of expression, conceptions, and ideas, although awaking each other mutually and associated most intimately, nevertheless preserve in relation to one another a certain autonomy.* Whether without words the *full* integrity, accuracy, and fruitfulness of thought can persist, is another question to which at present no unqualified answer in the affirmative can be given, in view of the observations just quoted from Lordat and Broadbent. We shall return to this subject again in Chap. 25.

CHAPTER VIII.

Words as Symbols, and the Facultas Signatrix.—Finkelnburg's Asymbolia, or Asemia and its Forms.—Relation of Aphasia to Asemia.

Imitation, by delineation, painting, and sculpture, of things perceived around us, contributes very largely to engrave sensory intuitions upon the memory. A Napoleon or a Frederick the Great lives before the inner eye of later generations precisely as his picture has represented him.

What the *drawing is for the intuition the symbol is for the conception*, and it is gained from the drawing by a kind of abstraction, as the conception is from the intuition. It fixes the conception upon the memory, and is so closely united with it, that this former is awakened by the symbol, and, conversely, the symbol by the conception. If we reduce a picture to a few striking, *i. e.*, essential outlines, the imagination immediately fills it out again to a picture such as existed before. But the imagination can do far more: the child makes a doll out of any or every piece of stick or stone in its way; the peasant is reminded by a roadside cross of some fatal accident, and also to pray for the poor soul of the departed; we tie a knot on our handkerchief to recall to our mind, by the sight of it, some promise or intended business. Thus, according to fancy, we may awake in our memory, by this or that sensory phenomenon, this or that conception, or a whole comprehensive series of them; we create voluntarily an intimate relationship between both, an intelligent bond by means of which one draws the other after it. The words of the phonetic language of to-day have hardly anything any longer in common with the imitative sound-symbols of primæval man, and only by the dictionary are we able to determine with certainty what is the meaning of this or that word of a foreign people. They have become phonetic symbols, whose association with this or that conception appears to be purely accidental. And, according to the principle of sound-metaphor, we may exchange visible symbols for audible, or the reverse. Phonetic writing (*sic*, phonetische Schrift) has clearly been developed from pictorial writing and hieroglyphs, and we are able at will to associate with the spoken, as with the written word, the same conception.

Now, apart from the symbols *which each individual makes for his own special requirements*, and from *the letters and words of phonetic gesture and written speech*, there are a multitude of others in use which the human race has gradually created, and which one generation has handed over to another: such are, *social* and *devotional forms of expression*, *cyphers*, *algebraical formulæ*, *geometrical figures*, *musical notes*, and so on.

These symbols are the *current coin of the collective intellectual intercourse* between individuals and nations; they represent, whether as words, artistic compositions, natural formulæ, and so on, the aggregate value of the intellectual property of humanity. Moreover, we often even run a risk of confounding our actual property with the symbols of value current for it, and accept words for ideas, *façons de parler* for true expressions, the form for the thing, cultivation for religion, the idol for the Deity. Savage peoples even go so far as to identify the name of an individual with the latter himself.[1]

From their neat and well-defined form symbols facilitate very materially rapidity of comprehension. An expression of face, a sound, a single word, often make a whole situation clear to us. We simply stigmatize a man as a bigot, as blasé, etc., and at once his whole feeling, thinking, and acting stand out before us as though illuminated electrically. The eloquence of numbers is proverbial. And the more abstract and exact thought becomes, the more it is elevated out of popular into scientific regions, the shorter and sharper do the symbols become; words are no longer adequate for the task of expression, and in logic, mathematics, physics, chemistry, etc., the most mature products of thought are consigned to algebraical formulæ.

But it would be an error to suppose that in symbols there was nothing but a means of communication with others. They are far more; they are *a component part and an essential keystone of all mental operations.* No thought has attained to thorough clearness and accuracy until it strikes home in the form of word or sentence. How long do we frequently torment ourselves over the solution of a problem! At last the proper word or the true formula suggests itself, and we give vent to our satisfaction in a joyous eureka. Words, to use a happy simile of Steinthal, are as footprints which the mind leaves behind when it traverses the paths of thought in search of cognition, and by whose help alone it is able to make its way with certainty.

The necessity for symbols in intelligent thinking is dependent on the *narrowness of our consciousness*, through whose focus intuitions and conceptions can only pass in linear succession, or in other words, in single file. Hence, we are only able to think rapidly by thinking abstractly, and only accurately when the abstractions acquire intuitive perceptibility in a neat, comprehensive form. The idea in verbal form fulfils these requirements.

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[1] Conf. the remarkable facts bearing on this noticed by *Tylor* ("Forschungen über die Urgeschichte der Menschheit." *Mueller*, Trans. Leipzig. Cap. 5 and 6); also *Lazarus*, "Das Leben der Seele." Bd. 2. 1857. "Geist und Sprache." S. 77 Anm.

Without the word forest we should, like the child, see trees upon trees, and again trees, but for very trees see no forest; but with the word forest the abstract idea stands before us as a clean-cut, comprehensive symbol, with which thought can deal comfortably in its further progress.

Finkelnburg[1] refers the capability of forming words to the general capability of creating signs and symbols. He insists that speech is to be regarded as a product of a special *symbolic function of the mind*, the *facultas signatrix* of Kant. He supposes this capacity to exist also among animals who are able to render their wishes known by means of signs intentionally or unintentionally, and to construe the signs of other animals, and even of man, correctly.

But if we regard the question more closely, this symbolic function will be seen not to be referrible to a special symbolic power of the mind. The symbolic capacity is nothing more than the *impulse which produces movements of expression, and the power of understanding these, and of utilizing them for the purposes of intelligent intercourse.* It consists in feelings, intuitions and conceptions which are most intimately associated with one another, and set in motion certain reflex mechanisms. That expressions are understood and acquire significance depends partly upon the memory, partly upon the associative mechanisms of the brain through which it becomes possible for feelings, intuitions, and conceptions to enter into orderly combination and evoke each other, mutually following a regular method. The organic mechanism on the one hand, and the force of habit on the other, govern the laws of this association. The symbolic function is a form of activity of the instinct and intelligence, differing from others only in the *end to which it is directed*, namely, *the seeking to comprehend and to be comprehended;* moreover it is, like the first, *the resultant of feelings, intuitions, and conceptions on the one hand, and of associative and reflex mechanisms on the other.* If the *facultas signatrix* of animals is not equal to the task of forming actual words, the cause is to be found solely in the rudimentary development of their intellectual capacity, and of the brute brain as an organ of thought. The animal does not speak simply because it lacks an *intelligence and impulse.* But our children begin to speak early, because they are born with this intelligence and impulse, because our brains have acquired in the long course of development of the human race the predisposing arrangement thereto. That it is really an unconscious and irresistible impulse which first compels children to speak is a matter of daily observation. It is only subsequently and by degrees that this impulse is subjected to the directing power of the will and the dictates of custom and reason.

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[1] Berl. klin. Wochenschrift. 1870. Nos. 37, 38.

This view of ours, then, of the symbolic capacity, is not compatible with the theory of the existence of a *special organ* for it in the sensorium. It is bound up on the one hand with memory, and on the other with the aggregate of the associative and reflex apparatuses of the organ of thought, combined for movements of expression. We do not mean to say, of course, that this aggregate of individual apparatuses may not become diseased in its several parts. Partial lesions of the organ of thought must be followed by partial damage to the symbolic function.

In fact, we find partial disturbance of the symbolic function, whether it be the memory that is obliterated or interfered with, or this or that association of conception-groups, or feelings, or this or that connection of sensory or motor tracts with the reflex centres. But suppose these become disordered or are even destroyed as a whole, then the entire symbolic function also suffers decided injury, even to complete annihilation. With Finkelnburg, then, we may, not incorrectly, from a symptomatological standpoint, speak not only of *asymbolia*, but also distinguish between a *partial* and *general form* of the same.

We do not here mean to infer, of course, that the symbolic organs can only serve symbolic purposes and not be otherwise employed. It depends entirely upon what associations and what reflex combinations are set agoing by impulse or will as to what different objects will be accomplished by the same organic instrument. We can use the tongue both for eating and speaking, and may be impelled to the same movements of expression by different feelings and conceptions. One lowers his head in humility, another from fear, a third from politic calculation, a fourth in sign of acquiescence.

Now, according as these or those feelings, conceptions, or reflex connections, and as the memory for this or that intuition and conception are disturbed, we may observe different forms of asymbolia. Thus during sickness we sometimes see the power of forming words by phonetic symbols alone disturbed, while they can still be expressed by written; but, as a rule, the loss of both is impossible, or only certain words and classes of words are lost. There are, according to Steinthal, ἄμουσοι, who, having lost the words of a song, are unable to recall the notes, while there are others who only lose the words, but retain their sense for the notes. Finally, many even lose the power of mimic expression together with that of phonetic and written language. These last, of course, have suffered severe losses in their collective mental property.

That condition in which persons lose the power of speech partially or completely, without their mind being involved, and without either a mechanical hindrance in the external apparatus of speech or paralysis or spasm of muscles or injury of the nervous structures which preside over the articulation of the several sounds, is known to pathologists either by the name *aphemia* (Broca), or better, by that of *aphasia* (Trousseau). Where, under similar conditions, there is no loss of speech, but a wrong word is substituted for the one meant, the state is spoken of as *paraphasia*. Commonly, however, the term *aphasia* is allowed to include in its wider sense that of paraphasia and to embrace also those conditions in which patients are senseless but make use of constantly recurring sound-symbols as expressions. Appearing under corresponding conditions, disturbances in the writing powers are characterized either as *agraphia* or *paragraphia*. With all these the faculty of understanding both phonetic and written symbols may either be destroyed or remain intact. It may be obliterated, although both hearing and sight are unimpaired. Comprehension of written symbols is far more frequently lost by aphasic patients than that of spoken words, and it has been called *alexia*. Here also, when there is a transposing of the written words, we may speak of a *paralexia*. Analogously one might distinguish between *amimia* and *paramimia*, when patients are unable to execute the characteristic gestures and expressions of face, or transpose them, as, for instance, when they make an affirmative instead of a negative gesture, or the reverse, as has been observed.

Finally, it has come to this at the present day, that under aphasia we no longer understand merely the disturbances in speech alone, but also the collective *symptomatic phenomena*, whether abundant or scanty, *under which the execution or comprehension of any given signs by which it is sought to communicate conceptions or feelings is impaired* Thus a new species of sickness has been created which presents all the light but also all the dark sides of purely symptomatic species. Finkelnburg is certainly right in proposing instead of the term aphasia, which only has reference to phonetic symbols, a more comprehensive one, *asymbolia*, which would embrace all the numerous as well as different clinical forms of *disturbance in the formation and comprehension of symbols*. With Steinthal, however, we should prefer the term *asemia*, the idea contained in "symbol" being more restricted than that contained in "sign." Behind the symbol there is always an idea hidden, behind the sign often only a feeling. We might then speak of an *asemia verbalis*, *graphica*, or *mimica*, and of the varieties *as. paraphasica*, *paragraphica*, and *paramimica*, and, in the case of asemias of expression, of *as. expressiva*, or of perception of *as. perceptiva*, according as the power of forming or understanding signs is lost. But, of course, we gain nothing more by this than a more accurate terminology and a better point of view for observing the functional nature of the changing phenomena of the disturbances in question. It will be the duty of science in the future to discover the cerebral tracts and centres through which the formation and comprehension of the various signs, phonetic and written words, numbers, gestures, and so on, are accomplished. It will also have to ascertain the minute and gross disturbances in the organic mechanism out of which the numerous forms of asemia spring.

CHAPTER IX.

Speech as the Grammatical Moulding of the Material of Thought which has been perceived and logically and metaphysically elaborated—Speech as a Pathological Symptom and Object—Definition of Disturbances of Speech, according to their Nature, as Dyslalia, Dysarthria, Dysphasia, and Dyslogia or Dysphrasia.—Lalopathia and Logopathia or Logoneuroses.—The Centre of Speech a great Central Organic Mechanism.

Our conceptions of things are never adequate, but only contain our *subjective* judgment as to their nature, which varies considerably according to the age and stage of development of the individual and of his nation. With the strengthening of the sensory and intellectual auxiliaries of cognition there is an increase in the breadth and depth of our inner possessions, and our whole feeling, thinking, and desiring being becomes altered. Our conceptions consequently are not the products of the perception and judgment of things by an unvaried being; if so, they would necessarily be unvariable. *They are the products of a changing but always at any given moment definitely constituted being*, whose cognitions have arranged themselves into distinct groups and circles of conceptions, and whose feelings and desires are excited in this or that way by this or that idea.

The conceptions which we form of things about us are consequently nothing foreign, external to ourselves, not the things themselves with which we are so prone to confound them because we only understand things through conception. Moreover, they do not dwell of nature in us, but are merely the *substantial contents of our intellectual personality*. In forming them we receive them into ourselves as organic parts of our mental being; we assimilate them, or, to employ a psychological expression in the place of a physiological, we *perceive* them.

This *perception* through which we come into possession of our conceptions may be compared to chemical action. New and foreign conceptions which crowd upon us work with a force, which in its evidences has much resemblance to affinity. But the material of thought which they offer must, in the thinking mind, encounter allied elements in order to the proper march of the mental action. Of what use to the savage are the microscope and telescope, Aristotle and Euclid, so long as he is ignorant of his A B C, and the multiplication table? But if once the inner movement takes place it depends upon the already existing circles of conceptions what kind of molecular disturbance will be produced by the newly added elements, what old compounds will be decomposed or strengthened, and what new ones will be generated. At the same time, under all circumstances, feelings will be excited and rendered latent, just as chemical action sets free and renders latent heat.

This *substantial contents* of our cognition, acquired by perception, must be clearly distinguished from the *logical*, *metaphysical*, and *grammatical form* into which it is pressed by virtue of our mental organization. Everything that we become cognizant of by perception and thought must find its way through the mould of judgment and conclusion into the being, and there range itself under the category of cause and effect, object and property, time and place, etc., and find expression in the grammatical form of substantive, verb, adjective, etc., as subject, predicate, and so on. Through perception the individual, as a psychological unit, acquires the material of thought, while logic, metaphysics, and grammar give form to this material—logic during its reception into the being, metaphysics by its arrangement within the being, and, finally, grammar by the expression through the being. These processes of *perception* and of *logical*, *metaphysical*, and *grammatical moulding of the material of thought* are all accomplished autonomously, *i. e.*, independently of one another, although always dove-tailed one into the other, and leading the being to the one end, namely, cognition. Their autonomy is easily seen in these facts, namely, that we may arrive at wrong conceptions through the most faultless conclusions, when we start from faulty premises; that a knowledge of things is quite compatible with the grossest error in regard to the origin and nature of the same; finally, that the most flowery nonsense may be uttered in a perfectly correct form of speech. Through this autonomy alone is the so-called *objectivity* of our judgment, the *accuracy of our cognition*, guaranteed. *A thought* *is true when it has fully withstood the triple fire-test of logical dissection, of metaphysical synthesis, and of grammatical expression.*

Speech, then, as the expression of thought, must be, on the one hand, capable of representing the matter thought, and on the other, its logical and metaphysical form. The child's earliest words and the word-roots of our national languages, are so to speak, but the *protoplasm of speech.* A root is not yet a substantive, a verb, or an adverb, or indeed a part of speech at all, but only a germ of such. It is nothing more than the primal form in which the first very general predicative or demonstrative judgments as to things are laid down; and only through the placing of these primordial words in relation one to the other, by emphasizing them, by their amalgamation, accentuation, and so on, is there gradually formed from such roots a language *organized out of parts of speech* and *grammatically suited* to all logical and metaphysical requirements. The mode in which this development takes place varies remarkably according to the conditions of different peoples.

Now, if speech be the faithful mirror of the human mind, as it is constituted under the endlessly varying inner and outer conditions of its development among nations and individuals, it must also faithfully reflect the morbid disturbances of the same. The perverted conceptions of delirium, the wild whirlwind of thought of mania, the slow and torpid flow of ideas of idiocy, all find expression through it. In all these cases speech is for us only *a symptom of the processes in the regions of perceptive activity; it becomes itself a pathological object* when it is disturbed as an *autonomous process.*

As an autonomous process, speech consists in *articulation* and *diction.* The first is executed as a motor act by the external organs of speech and the peripheral and central nervous apparatuses through which the multifarious co-ordinated inner and outer movements take place, whose products are sounds, syllables, and words. All lesions of articulation may be styled *dysarthric* disturbances of speech. But it is usual to designate those which depend clearly upon gross mechanical defects in the external apparatuses of speech and their motor nerves as *dyslalia* in contradistinction to the true or *central dysarthroses*, which are either produced by organic lesions or are only functional.

Diction is a mixed *senso-intellectual act,* through which words are not alone combined with conceptions as sensory symbols, but are also grammatically formed and syntactically arranged in order to give expression to the flow of thoughts. Disturbance of diction may be called *dysphasia.*

Dysarthric and dysphasic disturbances taken together may consequently be considered as constituting what are properly considered as true disturbances of speech, and may be combined under the name of *lalopathia.* The latter then includes *dysarthrias* (including dyslalias) and *dysphasias.* In all these disturbances it is only the question of a defect in the purely formal expression of the movements of thought in sounds, syllables, words, and sentences, quite apart from the *substantial contents.* The thought may be faulty, but expressed in an irreproachable form. Conversely a correct thought may be defectively expressed, but as soon as the formation of thoughts is disturbed it becomes a question of *dyslogia* and *logopathy*, or, as some would say, of *logoneurosis*, since every movement of thought takes place through the nerve-substance. The two ideas of logoneurosis and lalopathy consequently do not cover each other.

Now we have set out with the intention of subjecting chiefly the dysarthrias and dysphasias to a scientific analysis, and the dyslogias only so far as they lead to disturbances in talking, or *dysphrasias.* Otherwise we should be obliged to include in our inquiry the whole question of mental disturbances. We shall be engaged consequently on a pathological territory lying between the psychoses and the sensibility and motility neuroses, and intimately interwoven with all these, and at the same time connected, through the mechanical dyslalias, with that domain usually relegated to surgery.

For the purposes of speech there exists an apparatus as vast as it is complicated, consisting of nervous tracts and ganglionic centres which partly occupy the position of the loftiest workshops of the conscious intelligence and of the will, and are partly reflex agencies in which simple and ordered sensory stimuli are converted into motion. Such a thing as a simple "*centre of language*" or "*seat of speech*" does not exist in the brain, any more than a "seat of the soul" in a simple centre. The central organ of speech is, on the contrary, rather composed of a *large number of ganglionic apparatuses, widely separated from one another, but connected by numerous tracts, and fulfilling certain intellectual, sensory, and motor functions.* But it is probable that none of these apparatuses subserve alone the objects of speech. The nervous mechanisms can be made serviceable for different purposes, and it is only practice which brings about those connections between ganglion cell and ganglion cell, between ganglionic centre and ganglionic centre, which render speech, in its more restricted sense, possible, as well as all the other so very numerous means of expressing our thoughts and feelings. In this sense alone is a central organ of speech gradually *cultivated*, or, as some will say, created, in the brain by language itself, and in this sense do there also exist central organs for the plastic art, for painting, music, dancing, and for the forms of thought which do not give employment to words, but to numerical signs and other pictorial formulæ.

CHAPTER X.

Speech as a Product of Instruction, of Practice, and of Habit.—Memory a Fundamental Power of the Nervous System.—Its most general Vital Conditions.—Amnesia Totalis and Partialis.—The Historical Aspect of Speech Amnesia.

Instruction is the agent by which we place our children in the possession of our rich inheritance of speech. We teach them to utter their feelings and thoughts exactly in those forms of expression suited to the spirit, the whole nature of our nationality, the time we live in, and our position in society. From us they learn how to control their expressions and gestures, to clothe them in the mode prescribed by custom and fashion, to choose from the treasury of words the most current coin, and to adopt the rules of grammar. By degrees many strange sounds disappear, through which the child's first impulse to speak manifested itself—many of the sounds expressive of coaxing and of various emotions, through which mother and child at first conveyed their feelings to one another, and also the onomatopoëtic utterances characterizing the different objects of the animal kingdom; in short, the *natural language* of the child is overwhelmed by the *transmitted language of the nation.* This tremendous change is the result of *practice* and *habit.*

It is *practice* which creates the bond through which mental, sensory, and motor centres are united for the purposes of speech. What we call practice is not merely manifested outwardly in the muscles, joints, and peripheral nerves; of far more importance than the constantly repeated visible movements of the tongue, the lips, the eyes, etc., are the invisible, which, in the case of phonetic speech, are carried out in the brain, and, in the case of written and gesture language, in the spinal cord.

Practice has two great results: it not only teaches how to choose properly the muscles which are responsible for the attainment of definite objects, but it also substitutes for the originally orderless and useless expenditure of force and explosive discharge, a regulated use of the motor forces as economical as it is suitable. The kicking and struggling of a restless infant are gradually converted into grasping, seizing, toddling, etc.; the clucking with the tongue, hissing, sputtering, and lisping, into articulated words. Among the Hottentots and Kaffres alone have the sounds of clucking with the tongue been preserved in their alphabet.

Again, if we associate a definite feeling or conception with any given movements for a certain number of times, the latter will eventually take place involuntarily as soon as that feeling or conception is aroused, or conversely. Certain musical scores awaken in our memories certain words, or the words the notes, and we perhaps sing or whistle them softly to ourselves. The bond which secures to our central organs facility of communication between different stations of sensation, conception, and movement, is known as *habit.* Stations which are accustomed to correspond with one another answer each other's despatches very promptly, whereas they do not answer those from others, or do so only with hesitation or uncertainty. It would appear as though stimuli, when repeatedly transmitted from one point to another, were able to push aside hindrances all along the connecting tracts over which they pass, and to make the way freer, smoother, and more easily traversed.

Comparative philology furnishes numerous examples of the significance of practice and habit. There exist whole nations, or single branches of nations, to whom the pronunciation of the letters r, l, h, or ch, or of the dental th, or of diphthongs and other letters, offers the greatest difficulty, although they are by no means lacking in the necessary organs for their articulation. And as with the co-ordination of the articulative central stations, so is it also with the association of conception with conception, and their union with feelings and desires—they are all under the sway of education and habit. How utterly different are the thoughts, feelings, and wishes excited by the exquisite muscular development of an Antinous in the breast of a Winckelmann, full of ideal art, and those aroused in the head of a South Sea Islander, grown gray in cannibalism!

Closely allied to the principle of habit is the power of *memory*. The impressions which external things make upon our senses and intellect leave traces behind, just as the movements which spring from them. The more frequently the same impression is freshened up, the clearer and more lasting are these traces. They may be compared to the invisible pictures which the sun impresses upon a prepared silver surface. Just as these are charmed into being by the art of the photographer, so are the "picture-residues" of our senses by allied or analogous images, conceptions, and movements. In sleep such dream-images often follow each other loosely and as though accidentally in a panorama, excited, it is supposed, by stimulation of the central organs through the blood.

What we call memory really consists of two processes: (1) the *fixing* of perfected impressions in the form of permanent traces, the residua of stimuli, which remain behind as sensations, images, conceptions and motor acts (words, grasps of the hand etc., etc.)—this is *memory* in its narrower sense; (2) *the calling up again* of these different residua of earlier impressions from where they are stored, which is known as *recollection.*

Recollection takes place through processes of *association*, which may be referred physiologically to the conducting and transference of stimuli from one point of the nervous system to another, according to the laws of *reflex*, *co-movement*, and *co-sensation*.

We believe that it is something more than a mere simile when we speak of recollections being the residua of impressions. We hold the view seriously that, parallel with the sensory impressions and their residua, there are material changes in the nervous system which the external stimuli produce as they traverse the conducting filaments from nerve-cell to nerve-cell. If, for every separate sensation and movement, distinct peripheral and central receptive points and conductive tracts for stimuli exist, there must be for every picture, as it is compounded of many distinctly separate sensations, a corresponding distinct combination of filaments and cells; also for every motor act and every conception arising out of images and motor acts. Finally, there must be created just as many concatenations of nerve-cells as there are feelings, pictures, and conceptions stored up in the memory. Without the hypothesis of some material alteration in the nerve-substance, it would be impossible to conceive why the inclination to many movements originally learned, and consequently at first acquired, *should be transmitted to descendants*,[1] or why certain intellectual and artistic talents should be inherited from individual to individual in certain nations and families.

Therefore, with Ewald Hering and Laycock,[2] we must refer back the memory to that *general principle which preserves the type of the species in the organic world through countless generations, and secures the transmission of bodily and mental peculiarities* from the ancestors to the latest descendant. To this Laycock has given the name *synetic principle*. It is very closely connected with the commonest laws of development and degeneration of the organic and intellectual life. In receiving and retaining impressions the latter introduce a further series of organic and psychical processes which exercise an influence upon our whole being, changing and further moulding it, just as the impregnation of an ovum with this or that semen influences the future bodily and mental constitution of the individual to be developed therefrom.[1]

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[1] Conf. *Ch. Darwin*, The Expression of the Emotions in Man and the Lower Animals. *Darwin* cites a number of interesting examples of such hereditary acquirements.

[2] *Th. Laycock*, On Certain Organic Disorders and Defects of Memory. Edinb. Med. Journal, Ap., 1874. The same thought may be found excellently worked out in a lecture by *Ewald Hering*, Ueber das Gedächtniss als eine allgemeine Function der Materie, delivered before the k. Acad. d. Wissensch., 30 May, 1870, k. k. Staatsdruckerei, 1870. Conf. also an excellent article only known to me since the conclusion of my work: Das Unbewusste vom Standpunkt der Physiologie und Descendenztheorie. Berlin. 1872.

The old view must of course be abandoned, which regards the memory as a special receptacle in the brain or in the soul, in which images and ideas are stored up and arranged as if in pigeon-holes. As we hold, memory is a *fundamental power* of the nervous system, bound up with the elementary apparatuses of nerve-cells and filaments, and dependent in its achievements upon its partly congenital, partly acquired concatenation in the central organs. We also believe in the existence of memory among the senses, as well as in the intellectual and motor centres. Long ago Henle[2] was led to the belief in a *sensation memory* by the consideration of the images retained by the retina. It is really hard to see why a remembered bell-sound should not ring on in the same spot in which it rang while the bell was still vibrating, and where it still echoed some time after the latter had ceased to give forth a sound. Bain,[3] from whom we borrow this remark, is quite right in this sense when he designates the recollection of a saying as "suppressed speech," "ready every moment to pass over into uttered words." When we recall a verse of a song, or a saying, it is not alone the thoughts which present themselves, but also the words as sound-pictures and remembered movements, and these are even accompanied by slight movement, and not unfrequently by articulate action of the tongue.

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[1] These thoughts have been further worked out by *Laycock*, especially in regard to the retrogression of the psychical life to earlier stages of development, in a very thoughtful article, "A Chapter on some Organic Laws of Ancestral Memory," Journ. of Mental Science, July, 1875.

[2] *Caspar's* Wochenschrift. 1858. Nr. 19. Also Allgem. Anatomie. 1841. S. 739 f. Conf. also the thoughtful remarks on memory and sense-memory, by *Draper*, History of the Conflict between Religion and Science. New York, 1873.

[3] Geist und Körper. Leipzig. 1874. S. 109.

The commonest vital requirements of memory are :

1. *Supply of the nerves with adequate nutritive material.* Exhaustion from lack of food, overstrain either of the muscular or mental powers, weaken the memory.

2. *Proper distribution of the blood dependent on the structure and innervation of the blood-vessels. Senile amnesia* is an instance of the influence of the first item, *transient loss of memory due to plethora, etc.*, of the second.

3. *Proper condition of the organic soil which takes up nutritive materials from the blood.* Memory is dependent upon the congenital and acquired conditions of the brain, the greatness of its nutritive energy and developmental capacity.

4. *The necessary accumulation of assimilated pabulum, which is chiefly provided for by sleep.* Wakefulness at night weakens the memory.

If the memory is a *common function of the nervous system*,[1] the nature of its contents of any moment must depend upon the functional capacity of the latter, which varies according to the grade of development of the organism. The memory of man differs from that of animals chiefly in the intellectual association of what is remembered with the abstract idea of the time.

When memory is thus viewed, it no longer appears wonderful that it is only partially lost, as a rule, after cerebral affections. Sometimes only optic images, sometimes only acoustic, disappear ; now this, now that conception, or single words and knacks ; indeed, even words themselves are obliterated, sometimes as a conceptions, sometimes as sound-images, and sometimes as motor acts capable of being remembered. It is also less remarkable, then, that one man's memory is principally developed for numbers or formulæ, another for names of persons or places, a third for melodies, a fourth for verse.

We are justified, then, in speaking of *amnesia totalis* and *partialis* in a twofold but overlapping sense : in the first place, in reference to the *matter contained* in the memory ; and secondly, in regard to the *extent* to which the disturbance of memory is spread over the brain. Total loss of the cerebral memory must reduce an individual to the simplest condition of congenital reflex activity. He relapses into a state of animal stupidity, during which only breathing, the cardiac movements, swallowing, and such like, take place. But it is only very severe affections, involving the whole brain, which produce such consequences. It is the custom, however, to characterize also as complete loss of memory those disturbances in which the intelligence has become a *tabula rasa*, but in which many acquired forms of movements, such as walking, grasping food, etc., which involve a cerebral memory, can still be performed. Here the *sensory* or *instinctive memory* remains, and these movements can be executed, although only in a clumsy way; the *intellectual memory*, on the other hand, is destroyed, and intelligent speech is impossible for that reason, or, at the very most, some senseless sounds and sound-compounds are uttered. In such instances we usually expect to find morbid processes diffused over the whole cortex of the cerebrum, while the deeper ganglionic centres of the corpora striata, optic thalami, corpora quadrigemina, etc., still perform their functions. In partial loss of memory of the intelligence and speech the cause may be twofold: either there exist more or less trifling diffused lesions of the cerebral cortex through which only the looser and not the firmer functional concatenations of ganglion-cells are jarred or broken, or there are severe circumscribed injuries of the cortical substance by which the sensory or motor circles which close these or those chains of conceptions of words, are ruptured.

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[1] Ueber Gedächtniss des Rückenmarks, conf. *Freusberg*, Pflüger's Archiv. Bd. X. 4, 5.

That the memories of early days are less easily lost than those of a riper age, in cases where the powers fail in advanced life (*amnesia senilis*), is easy to understand. Not only is the brain in youth more capable of receiving impressions than in old age, but the recollections of that time will pass far more frequently across the mind than those of later years.

Even the physicians of early classic times had noticed the loss of memory for words through sickness and injury—Hippokrates, Galenus (in several places), Pliny (Hist. natur. Lib. VII. § 20), and others. Schenkius (Obs. med. Lib. VII. p. 480. Lugduni, 1585) was the first to notice the connection of loss of speech and of memory for words with affections of the brain, and that loss of speech could exist without paralysis of the tongue. Gesner (Sammlungen von Beobachtungen in der Arzneigelehrtheit, Nördl., 1772. II. 107), was the first to describe aphasia under the name of *speech amnesia*, and Alex. Crichton (An inquiry into the Nature and Origin of Mental Derangement. London, 1798. T. I. p. 337), under the title Diseases of the Memory, as a special disturbance of the memory. Most physicians of the last and beginning of this century grouped all the forms of loss of speech together under the name *alalia*, and dealt with it together with aphonia, from which they only gradually learned to separate it. The expression alalia is used by Delius as early as 1775, in an article "De alalia et aphonia (Nova acta naturæ curiosorum. T. VII. obs. XVIII. Norimb., 1757). According to Hammond (New York Med. Record, March 1, 1871. p. 1), an American physician, Rush, was the first to distinguish between different forms of aphasic disturbances of speech in the following rather rough way: 1. Forgetting of names and words of every kind. 2. Forgetting of names and words, with substitution of others totally different, which have no relation to the first. 3. Forgetting the name for a thing in the mother-tongue while the name for it in a strange or dead language is easily found. 4. A converse relation to No. 3. Dr. Scandella, an Italian, who died in New York in 1789, spoke during his illness first only English, then only French, and, on the day of his death, only Italian. 5. Forgetting of words, but not of the letters composing them. A case of a clergyman is quoted, who was obliged to spell words in order to render himself intelligible. 6. Forgetting how the commonest words are spelled. 7. Forgetting names and ideas, but not numbers.

CHAPTER XI.

Co-ordination of Movements.—Impelling and Restraining Forces.—Goltz's Croaking Experiment.

The ganglionic centres of which the nervous system is composed are as much registering as regulating apparatuses. They enable us to recognize external things in the form of images and conceptions, which leave behind their impressions, and to give expression to this cognition by indicative movements suitable to the images and conceptions. What we call *co-ordination of movements* is always accomplished by the preformed central anatomical mechanisms according to the laws of conduction, collection, and transference of stimuli. But the functional combination of the ganglion-cells in these centres is only in part existent *at birth*, as for instance, that for breathing, the cardiac movements, swallowing, etc., and that for the instinctive cleverness of animals which is nowadays regarded as inherited from progenitors who had first to acquire it themselves. Most of our human movements, however, are learned *by practice.*

But, however co-ordination is originally provided for, we find stimuli acting at one time with *impellent*, at another with *restraining force.* Running parallel with all reflex and voluntary acts there are constantly actual and suppressed movements. It is not yet ascertained whether there exist special motor impellent and special restraining mechanisms, but it appears probable that the same apparatuses provide for both impulse and check, and that it is merely a question of the measure, origin and direction of the stimulus influencing them, what result follows.

The celebrated *croaking experiment* of Goltz throws very considerable light upon the varying play of impellent and restraining forces upon the ganglion-cells functionally combined for co-ordinated movements of expression. Here we see that in the respiratory and voice centre there is a bond established between the ganglion-cells of the respiratory and vocal apparatuses, and that this "*croaking centre*" may be roused to activity at one moment, or checked at another, according to the wish of the experimenting physiologist. This, to be sure, the latter can only bring about by depriving the frog of that organ which gives this very lowly organized animal the power of manifesting its will clearly in relation to the human will. As long as the frog possesses a brain it does not, as a rule, croak whether we stroke its back much or little. As soon, however, as the cerebrum is cut away above the corpora quadrigemina, every soft stroking of its back evokes a croak. But if one of its legs be now pinched or ligatured, no amount of stroking will call forth a croak. By a roundabout way, however, we may even cause a frog with an uninjured brain to croak or remain silent, just as we will. If, for instance, we place him on a warm summer evening in his native swamp and among his kind, he will very soon commence to utter the well-known note expressive of quiet enjoyment; but if, after a moment, we throw a stone into the water near him, he will cease with the rest to give forth any sound.

Far be it from us to compare the miserable will of a frog with the glorious will of man, but the physiological principle of movement springing out of feelings and motives holds good in regard to the brain of the frog as much as to that of man, only that everything in the former is far simpler than in the latter. Conditions of cerebral excitation causing a feeling of comfort and enjoyment lead a frog to croak with unvarying monotony on one evening as on another. The same condition of cerebral excitation in man draws from his organs of voice the most diverse forms of joyful song or pleasant words of banter; but if sad news be announced, both song and joke are silenced until perhaps a cheering word is spoken in the circle and the same joyful frame of mind is restored.

All human education tends to the control of the congenital and acquired reflexes by intelligent and rational motives. The inner impellent and restraining processes withdraw themselves from our observation. The external means are training by admonition, example, and punishment, by motives of policy, equity, morality, and many others. The schools, the state, and the church emulate each other in modifying our natural reflexes. Up into the highest spheres of free will we may trace the constant struggle between impellent and restraining forces, between low but strong, sensuous and high ideal but slowly penetrating motives. The will which creates free men, heroes, and martyrs, grows strong by degrees only, for our emotions and impulses are constantly threatening to break through all restraint, like wild beasts. Sometimes the intuition of a movement, aye, or even the conception merely of it, will allure from the recesses within us reflex gestures, expressions of feature, words and sentences, to our great dismay and sometimes prejudice. And in cases where feelings and desires gain the upper hand through morbid excitation, and wild mania sits supreme, then all the barriers of will are broken down and speech unrestrained becomes a toy of the storms of emotional feeling.

CHAPTER XII.

Speech and Consciousness.—Deliberation.—Readiness of Speech.

The intellectual contents of our being slumbers unconscious in our memory until thrown into vibration by some vigorous shock from without or within. The vibrations, however, take place in such a manner that out of the whole undulating sea of thought only the highest waves reach the *visual field of the consciousness* (Wundt), all the remainder moving on unconsciously in hidden deeps. And even the loftier waves which do reach the visual field of our mind are not all presented to us at the same time with equal distinctness. Wave after wave, as it traverses the field of vision, is always only perceived in one point alone, the *mental focal point*, with all the vividness of perfected consciousness.

Steinthal proposes to term as "vibrating" all those *unconscious conceptions called into motion* within the deeper recesses of our being, in the discovery of which subsequently an analyzing judgment versed in the secrets of psychological life may sometimes, but not always, be successful. These he contrasts, on the one hand, with the *motionless unconscious* conceptions asleep and hidden in the memory, and on the other, with the *conscious*, those which to a certain extent visibly give the start to a whole series of movements. With equal right three conditions of sensation may be distinguished, and we may speak of motionless, vibrating, and conscious sensations. We shall come later on (Chap. XIX.) to the more detailed consideration of the relation of thinking and sensation to consciousness.

In speaking, reading, and writing there always run parallel with one another conscious and unconscious sensations and conceptions, and there is reason to believe that the mass of the merely vibrating is always in excess of the conscious. This holds good just as well for the operations of diction as it does, though to a greater extent, for those of articulation. Hence, it is not so wonderful as it would at first seem that individuals fully sensible of what they wish to say are yet unable to find the word they seek, or that paralyzed persons in uttering the words which they have learned as a whole, and not letter by letter, or according to syllables, cannot control such syllables or the several letters, some of which may be lacking or find their way into a wrong position or at a wrong moment, like a team of unruly horses.

Should impressions work sufficiently powerfully to give a shock to the whole being, the inner excitation must be reduced more or less in intensity in some way so as admit of *deliberation;* otherwise we have the phenomena before us described by the old poet in the words "vox faucibus hæsit." If merely single groups of conceptions of the being be called upon, the extent to which they were set in motion will reflect back with such force of feeling upon the being as to awaken its interest and produce a *readiness* to follow up the thoughts; and to clothe them in words will depend upon the age, sex, nation, religion, rank, culture, etc., of the individual. This is known as *readiness of speech*, and is a matter both of feeling and of the intelligence.

CHAPTER XIII.

Emotional Readiness of Speech.—Humor and Disposition.—Mental Readiness of Speech.—Collectedness.—The Speech of the Emotionally Disturbed and of the Insane.

Let us now examine more minutely the inner *preparatory* processes of speech which present themselves always either only partially or not at all to our consciousness, and are also only partially subject to our will.

The influence of the *state of the feelings* or the humor upon speech is well known. A pleasant event, an eventful day, or a *bon mot* spoken in the family circle, loosens the thoughts and the tongue, puts us in good humor and *disposed* to chat, to discourse, or to propose a toast. Thoughts and words present themselves to us as though by inspiration. On the other hand, vexatious events, depressing circumstances, the want of tact on the part of a friend, feelings of moral wretchedness, put us out of humor and render us *indisposed* to speech and check the current both of thoughts and words.

Intellectual preparation takes place by *collectedness* or *concentration* of the *attention.* If, for instance, we are absent, incapable of collecting ourselves on account of some unfortunate sensation or conception, both our thoughts and speech are embarrassed; we are in constant danger of making slips in sentences, words, syllables, and letters.

Perhaps a glass of wine or a cup of tea may put us into the *right humor* for speaking. They act like grease on an axle; thoughts and words roll forth smoothly and lightly in the boldest periods and most daring forms of speech, whereas just before we may have been struggling laboriously for a mode of presenting our thoughts. The same may be observed in the commencement of fever and in the earlier stages of *mania.* It is true the unwonted coloring and the warmth of expression may strike us, the motiveless exaltation of feeling appear strange and puzzling, as long as we do not recognize the sickness; but the material quality of the utterances and their rhetorical form cannot but be recognized as good, and even perhaps more than usually appropriate. Then, with the increase of the sickness and excitement, confused images and wild flights of ideas take the place of the bold, but, with all their rapidity, still orderly trains of images and thoughts, and we have *polyphrasia;* or, again, in the case of those of a lower order of intellect, with trivial minds, *talkativeness, or logorrhœa,* may regularly degenerate into senseless *lingual delirium.* Eventually it may come to this, that words and articulated sounds no longer suffice for the wild pressure of feeling in which the masses of conceptions in the maniac whirl along chaotically, and the patient shouts inarticulate sounds night and day; and even when the hoarse voice fails, the effort is still made.

Conversely, the *miseries of dyspepsia,* or those following the *abuse of alcohol,* may hinder lamentably the wheel-work of thinking and speaking. When an individual in such a condition is obliged to think out an idea or to deliver an already well-considered speech, he requires a far greater expenditure of force of will than usual, and a consequently almost painful strain on the brain, resulting in extreme fatigue of the latter ; and even so he only half attains his object. The thread threatens every moment to snap ; the proper word, presenting itself as if in mockery a moment before, vanishes the instant it is needed, and another, perhaps resembling it in sound, but differing in sense, forces itself on us, producing an unexpected contrast perhaps, which is greeted by the mischievous audience with peals of laughter. Then the speech drags on hopelessly, like a heavily laden wagon with worn-out axle-trees, on a rough road.

It is in a precisely similar manner that those sunk in *melancholy* experience a difficulty in speech at times almost painful. They answer slowly, speak low, in a tedious monotone, or tremulously, sometimes stop in the middle of a sentence or word, and repeat themselves. In some extreme cases of melancholy the patient may only prepare to speak, without really bringing out a sound, or in other cases the power to make even the attempt fails him, and he sits motionless, dumb, and brooding.

Just as humor is strongly influenced by morbid and unusual conditions of the body, so also is the mental readiness. If the whole being of a *maniac* be filled with wild ideas, his feelings, perceptions, desires, and conduct will be all determined by these, and voice and speech are changed, as his being itself is changed. The patient commands where he was before accustomed to obey, endeavors to teach where he formerly sought instruction, contradicts what he would at another time have affirmed. He speaks in the language of holy writ, declaims in rhyme and verses, mutilates foreign languages, or, mayhap, creates a new language for himself,[1] with new symbols and words for the new feelings and conceptions of his inner being. Again, he may employ old words and signs in another sense, rendering himself quite unintelligible. Occasionally it is only those perceptions and conceptions congruent with the mania which produce a perverted manner in an insane person ; so long as we do not touch upon his *idée fixe*, he speaks quietly and intelligently; but the moment we strike the keys which lead to the false chords in the scale of his ideas, nonsense bursts forth, and his demeanor, voice, gestures, and words are perverted at once.

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[1] Conf. *Snell*, Ueber die veränderte Sprachweise und die Bildung neuer Wörter und Ausdrücke im Wahnsinn. Allg. Zeitschrift für Psychiatrie. Bd. 9. 1852. S. 11. ff.

While in the deranged individual the being is only altered, the mental constitution differently conditioned, compounded of new elements of feeling and conception, which are still in complete readiness and capable of expression even with wit and acuteness (*folie raisonnante*), it is otherwise with the *demented*. Here the whole being is actually shaken in its constitution or quite fallen into decay, and the mental readiness is more or less annihilated, like the effectiveness of a routed army. Disordered, unconnected conceptions hurry hither and thither across the overclouded field of the consciousness, like the wreck of the shattered intellect.

In the *weak-minded*, finally, there is, to be sure, a personality still in existence, but a weak one, poorly furnished with only the coarsest sensuous feelings and scanty conceptions, only reacting readily to very intense impressions, and then merely with sluggish, dull, almost meaningless gestures and rough words; or, again, when it reacts easily, lapsing into fatuous, nonsensical, and often disconnected utterances. Lastly, in the complete blank, as regards emotion and thought, of the *wholly idiotic*, the capacity of expression is gradually completely extinguished; where nothing is any longer present, there also nothing can be placed in readiness any more.[1]

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[1] Conf. the handbooks of mental diseases, especially *J. Spielmann*, Diagnostik der Geisteskrankheiten. Wien, 1855. S. 26. 100. u. a. a. O. *Esquirol* (Traité des malad. ment. T. II. p. 288) distinguished between several kinds of mental weakness on the criterion of speech alone. In imbecility of the first stage, speech is free and fluent; in that of the second kind, less easy, and the fund of words poorer. In the first stage of true idiotcy, the idiot has only command over short words and sentences; in the second, only over one-syllable words; in the third, every articulate expression is lost.

CHAPTER XIV.

The Development of Articulation in the Child.—The Significance of Sounds in the Word.

The performance of speech is dependent on the power of articulation and diction. Let us then inquire more minutely into the inner mechanism of both acts, and, in the first place, of articulation.

By *articulation* we understand the sum total of the inner and outer movements, by which words as articulated sound-compounds are formed, independently of the ideas conveyed in them. The articulations of words are their *syllables*, which, however, are as a rule anything but the simplest elementary sounds: they are themselves compounded of *letters*, of *vowels*, and of *consonants*.

Now, as articulation is an acquired power and consists of co-ordinated movements mastered by practice, we must in the first place inquire *how we come into possession of it.* Three periods of development may be distinguished.

1. Within the first four months, and about the time at which the earliest movements of prehension commence, children when in a good humor frequently give vent to their feelings of joy in various sounds not prompted by those around them. This "lisping of infants" consists chiefly of lip-sounds and vowels, but also of linguals and palatal noises. We hear partly the well-known sounds of our alphabet, but not yet in the firm and clearly defined form which they assume later; and partly other strange noises almost incapable of being rendered in our letters, sounds of sputtering, hissing, growling, and clucking, etc., *e. g.*, pf, pfi, fbu, tl, dsi, qr, etc., etc. These are only accidentally and loosely combined with one another, as a rule.[1] These earliest utterances, which I may be allowed to call the *savage sounds*, are of purely *reflex* nature. They are the outcome of the same muscular impulse which leads infants to fumble about with their hands and kick with their feet, thus paving the way for prehension and

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[1] Conf. *Berth. Sigismund*, Kind und Welt. Braunschweig, 1856. S. 28.

walking. They may be regarded as those *primal sounds* with which man has been endowed from the commencement of his existence on earth, from which have been elaborated in the course of countless generations those other sounds which alphabets of the existing languages contain. It would be interesting, but would lead us too far, to inquire why different peoples have cultivated or rejected different sounds; why the Hottentots, for instance, have retained so many of the clumsy and hideous gutturals.

2. Later, when the child by listening learns to distinguish tones—at that time when it is learning to grasp things with the hands and to creep about with its feet, and at which the impulse towards imitation presents itself with its whole original force—these savage noises are gradually crowded out by the *conventional sounds of the national language*. It is probably due to an early awakening of the musical sense[1] that children take in and reproduce the vowels and diphthongs in those words which they hear before they imitate the consonants. This imitation, however, cannot be said to correspond to a comprehension of the words. They understand certain of them without imitating them, and imitate many without understanding them. The great difference between comprehension and conception, on the one hand, and articulation on the other, presents itself to us, therefore, as an object worthy of study even at the very commencement of the development of speech.

These earliest *clearly articulated sounds* are yet of a very simple kind. The child expresses its joy, amazement, dislike, and so on, with an a, aȧ, ho, u, da, etc., etc. These are pure *reflexes of feeling* or *interjections*. The *imitative sounds* are the well-known baba, bebe, dada, mamma, dodo, dudu, atta, etc., which nurses are over and over again repeating to their little charges. But at first the child does not connect any distinct idea with the words mamma and papa; it imitates the sound only as an acoustic picture, and only by degrees commences to intertwine the sense with it which the nurses have intended. It is not without interest to note that children seem to have a strong inclination to repeat syllables of similar sound two or more times over, or to combine syllables in inverted order, as adda, oddo, or, again, arrange one after another such as have a resemblance in ring, as bimbam, ticktack: this may probably depend upon the musical sense.

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[1] Sigismund's boy sang melodies with his father, and imitated melodies only in the higher octaves before he imitated words. (A. a. O. S. 118.)

The enjoyment of imitation awakes at different periods in different children, and their powers in this respect are just as various. At first the mimical words of children have only a very faint resemblance to those spoken around them, and are mostly only understood by the members of the child's family; but improvement takes place in this respect with ever increasing rapidity. Highly endowed children sometimes acquire considerable facility before the termination of the first year. Others again only manifest a pleasure in articulate speech in the second half of the second year, or even later, and then only make very slow progress.

3. At a third stage of its development the child learns to associate certain definite objects with the words acquired by practice, which are then gradually worked up into intelligent conceptions. Then for the first time speech becomes an *expression of thought*, interjection and onomatopoësis are elevated into true diction. It often occurs that a child, after using a word for a long time, suddenly becomes aware of its true significance, and that marvellous amalgamation of conception and verbal expression takes place, the commencement of *intelligent language*. At this stage for the first time the child creates out of itself, like the earliest inhabitants of the earth, certain onomatopoetica of things perceived which, however, must soon give place to the language of custom.[1] In all cases children display a greater readiness of

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[1] A boy of one and a half years, whom I observed almost daily, and who was just beginning to learn a few intelligent words, *e. g.*, papa, mama, hotto (for horse), and the demonstrative "da," which he accompanied with an indicative gesture of the finger, greeted every rolling object, balls, coins, balls of cotton, lead-pencils, etc., with the ejaculation "Golloh!" This is an intuition-reflex in the form of a sound-metaphor. "Goll" imitates not only the noise of a rolling body, but also rolling movement by a similar one of the tongue. The additional "oh" appears to be an exclamation of wonder. A step further and "golloh" might become as "goll" a root-word expressive of rolling movement. Conf. a similar observation by *Steinthal*, Abriss d. Sprachwissensch. 1871. S. 382. A girl of one and a half years imitated in this case all rolling movements with the exclamations "lululu," "bululu."

comprehension for the usual onomatopoetica of the nursery, as for instance, wauwau, miau, etc., than for the customary indicative expressions of the popular language. At this period the work of development of articulation proceeds uninterruptedly and hand in hand with that of diction.

From this description of the development of articulation in the child, it would appear that the human being at first produces simple sounds and combinations of sounds of a still indefinite and more or less accidental character, by a kind of inner impulse. He then learns to form these more accurately, and then passes gently upward from the simple to the compound, from the easy to the difficult, always admitting that there is a great difference in both individuals and peoples. What we articulate without difficulty offers an insurmountable obstacle to the South Sea Islander, whose language possesses a very limited fund of sounds.

It would appear that Steinthal's statement cannot be quite correct, that we never in our lives have learned single isolated sounds which we have combined later on in speaking, but that we have always listened to whole words. This holds good only for the acquirement of the popular language, and even here not completely. The fact of learning the popular language is always preceded by practice in the savage sounds and nursery talk, which only employs syllables and combinations of syllables, and constantly appeals in the most attractive way to the congenital musical and onomatopoëtic feeling. Later on an etymological and grammatical feeling springs up which is not congenital, but the result of education, and this in its turn takes the lead.

But that even in learning the popular language each sound and each syllable has its own special worth in verbal expression, is shown by the method which is found most successful in our public schools. Here children are made to break the words phonetically and to spell them and form them into syllables, in order that a correct pronunciation of the words and the component parts of each may be fixed on the memory. Later, we stamp on our memories the words of foreign idioms with more ease by pronouncing them aloud syllable by syllable. Moreover, it is a matter of experience that aphasic persons may be placed again in possession of their powers of speech by being taught to spell and form syllables.[1] Finally, the study of articulatory disturbances of speech teaches us that the formation of letters, their union into syllables, and the combination of syllables into words, are separate functions. These, although in speaking, as a rule, they go together, and although they are acquired at the same time, may nevertheless be separately affected by disease, and they possess consequently a mutual independence due to their different organic mechanism. It is just as in dancing, which consists of a series of elementary forms of movement, flexion, extension, abduction, adduction, bending, etc. These imply for each a special co-ordinative spinal centre, and they again may arrange themselves into higher movement units of ever increasing complexity (as, for instance, the separate figures of the "française") which are governed as to their extent and duration by higher co-ordinative centres situated in the brain. There is just this difference apparently, that in speaking we must only seek the lowest articulation centres in the basal ganglia of the brain, especially in the medulla oblongata and in the spinal cord, only so far as the centres of the formation of voice and respiration extend into it. For the sake of clearness let us glance for a moment at those *disturbances of articulation* more commonly observed.

Stammering consists in an incapacity to pronounce the letters properly, while in *stuttering* there is temporarily a spasmodic inability to vocalize certain sounds, especially the explosive consonants. Although the stutterer is able to produce all the letters separately, he is unable, on account of the difficulty of vocalizing them, to combine them all so as to form syllables. The stammerer suffers from *alalia* or *anarthria literalis*, the stutterer from *alalia* or *anarthria syllabaris spasmodica.*

In *stumbling over syllables* there is no more inability than in stuttering to form sounds correctly, nor is there want of power to vocalize consonants, but the stumbler so tosses the letters and syllables one over the other that distorted words are produced. Finally, the *atactically aphasic* person is able to form the sounds and syllables of words which his dictionary still contains, irreproachably, and they are frequently very difficult to articulate; but he is unable to group these sounds and syllables in any other way than just to form such solitary words as he retains of all his former store. He may say "kaffe," but not "keffa" or "feka" or "fake," as was the case with one of our patients.

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[1] Conf. an instructive case by *Schmidt* (Allg. Zeitsch. f. Psychiatrie. Bd. 27. S. 304). An aphasic patient was only able to comprehend written and spoken words when separated into their component letters and syllables, and learned to speak once more by spelling over and forming syllables. We give this case in Chapter XXVI.

There can be no doubt, then, that *the co-ordination of muscular movements to produce letters is a different function and connected with central apparatuses, distinct from those producing syllables and words.* It will be the task of the clinical physiologist to discover these different central apparatuses, for the experimentalist has no means here of elucidating the matter and can only, at the very most, give some little support to clinical observation.

Before we enter upon the question how far we are able at the present day to do justice to this very difficult question, let us inquire what are the sensory sources of co-ordinative excitation flowing towards the motor centres of speech.

CHAPTER XV.

The Sensory Reflex Sources of Phonetic Speech, and the Regulating Sensory Principle of Articulation.

It is customary to regard the hearing as both the *primary reflex source* and the *regulator of phonetic speech.* But this view as so expressed we must discard.

Laura Bridgeman,[1] whose history we have related above, lost her eyesight and hearing about the end of her second year when she was beginning to talk, and in consequence soon lost the power of speech. Now, although it may be assumed that she retained no memory of sounds, which long experience teaches us is the rule in the case of those who become deaf-mutes so early,[1] yet she was able to produce a multitude of sounds. She took the greatest pleasure in giving utterance to these, and even went so far occasionally, in order to indulge herself in noises to her heart's content, as to lock herself up when her teachers endeavored to restrain her in so doing. These were in fact inarticulate, *e. g.*, a kind of chuckling or grunting, as an expression of contentment, while others, *e. g.*, "ho-o-ph-ph," were better formed and served for an indication of astonishment. Other sounds, again, she elevated to the dignity of names and bestowed them on certain persons. She uttered these latter when those so denominated approached her, or when she wanted to find them, or even at times when she only thought of them. She employed some fifty or sixty such sounds as names, many of which could be written down, such as, fu, tu, pa, fif, pig, ts, pr, lutt, etc.; many, however, it would be almost impossible to express in letters. She formed, however, only words of one syllable, but frequently reduplicated them two or three times, as fu-fu-fu, tu-tu-tu.

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[1] *Fr. Lieber*, On the vocal sounds of *Laura Bridgeman*. Smithsonian Contributions. Washington. Vol. II., 1851, Art. II.

Here, then, we see how far an intelligent human being can advance in the acquirement of phonetic language *without either the power of hearing or that of seeing, and only through the medium of the muscular sense and that of touch.*[2] It is not only able to produce the "savage sounds" of the first stage of development of the lisping child, but is also able to register them in the memory, to combine them into orderly, although very simple monosyllabic sound-compounds, and to associate them with certain feelings, or even with conceptions of certain persons.

Deaf-mutes also, as Heinicke has pointed out, sometimes create for themselves, even without direction, a phonetic language, though a very imperfect one. This observer based his method of instructing deaf-mutes to speak aloud, for which he deserves such immense credit, partly upon this discovery. The sense of sight enables the deaf-mute to imitate the movements producing sound in the form presented by the teacher, and the regulating power of the sense of hearing is replaced by the muscular sense and that of touch.[1]

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[1] *Bonnafont* (Bull. de l'acad. de méd. T. XXX. p. 860), who has paid much attention to this point, assures us that children up to their eleventh year who become deaf from any sickness will soon likewise become dumb (even within half a year), and moreover, partially or completely so, according to the degree of sickness. He bases these conclusions on an observation of more than twenty cases.

[2] *Lieber* gives an account of three other deaf-mutes, far less intelligent than *Laura Bridgeman*, who never advanced beyond a few unpleasant sounds.

Samuel Heinicke believed also that he might call in the aid of the sense of taste to give precision and permanence to the sounds acquired by deaf-mutes, but his successors have abandoned this idea.

All that touch and muscular sense are able to accomplish under the direction of sight is shown in a most interesting way in the case of deaf-mutes who have been instructed. They learn to articulate and to speak correctly, though their voice is rough and unpleasant. But it is most astonishing to what extent some of them can acquire the power of articulation.[2]

Now, from these facts, the following conclusions may be drawn:

1. *That at all events hearing cannot be called the only primary reflex source of those sounds which we employ later in speaking in a permanent and distinctly articulate form.* The first lisping of the child springs from a feeling of enjoyment, just as the frog in the pond croaks from a sense of enjoyment. These pleasurable feelings working in the cerebrum cause a stimulus to the

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[1] There are, moreover, some apparently quite deaf and dumb persons who are great lovers of music and visitors of concerts, who can distinguish between well and ill executed passages, and who applaud or blame with correct judgment. It is possible, of course, here, that the vibrations of sound generate musical feelings in the acoustic apparatuses and centres by propagation through the bones of the skull. Compare, for instance, some interesting observations by *Mueller* (of Pforzheim), Allg. Zeitsch f. Psychiatrie. 1849. Bd. VI. S. 242. *Kruse*, an instructed deaf-mute, was able to distinguish the tones of different instruments. He compared those of the trumpet to a yellow color, those of an organ with green, those of a drum with red (*Tylor*). Conf. Chap. XXXVI.

[2] I conversed one day in hospital for a considerable time with a young bookbinder just admitted for phthisis, without noticing anything peculiar about his speech, except a loud voice and an unusual pedantical accuracy of expression. It was only on terminating my examination, after taking down his clinical history, that I noticed to my astonishment that I was conversing with an educated stone-deaf individual. Noticing him now more closely I found that be read every word rapidly and with certainty from my mouth, and that his speech was hard, *i. e.*, destitute of music and modulation.

basal centre of sound; but this is not the first point of origin. It is possibly a sense of satiety and of warmth proceeding from the gustatory and sentient nerves of the skin which gives rise to this feeling of comfort and to the muscular craving which impels to the utterance of sound.

2. *Hearing is not an indispensable regulator of articulation. This axiom, however, must be to a certain extent modified, for it is only by the co-operation of the hearing sense that delicacy and smoothness of articulation and euphony in speech are secured.*

3. *The power of imitating phonetic speech is dependent on hearing and sight; but only one of these senses is really an indispensable requirement in the matter.* Blindness and deafness combined, existing from earliest infancy, admit, indeed, of the acquirement of the rudiments of phonetic speech, but render its full development impossible, while in such a case a language of gestures and tangible letters and symbols may be learned.[1]

4. *The full development of the intelligence and the acquirement of a language involving ideas are always dependent, at the very least, on the sense of touch and muscular sense.* In a case of blindness and deafness combined these are essential and indispensable. For the power of properly estimating the resistance of the muscles to be set in motion by articulation and of overcoming this by a simply adequate force, is here just as indispensable as the other, namely, that of appreciating sensations as to the position of our members in space when engaged in movements. The first is provided for by the nerves of muscular sense (whose existence, established by Sachs, was felt to be such a necessity to physiology); the second by the tactile nerves.

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[1] I find in Boz (loc. cit. p. 74) the history of a boy, Oliver Caswell, who, like Laura Bridgeman, had become blind and a deaf-mute, and who was taught to read by Dr Howe.

CHAPTER XVI.

Imitative Articulation.—Is there a Direct Reflex Tract from the Acoustic Nerve to the Basal Centre of Sound, or does it lead through the Cerebrum?—Experiments of Vulpian and Danilewsky.—Persistence of Imitative Speech.—Loss of Voluntary Speech.

With the child an expressed word makes such a strong impression upon the sense of hearing as to impel to imitation, even when not understood. This reflex-motive power dwelling in the word itself as a perceived sound-picture, and quite independent of its inherent meaning, is seen in undiminished strength in the child, all reflex effects being particularly easily produced during childhood. But as the organs of intelligence are gradually developed, the ever-increasing processes of excitation in the cerebrum, and especially in the cortex, come into play, checking the mere sensuous reflexes. The adult only imitates when carried away by some unusual force in a word laying hold on his feelings, or with some design, or when from disease of the cortex of the brain the restraining influences over the sound-reflexes are to some extent interfered with. This will explain the "echo speech" of many persons who have become mentally weak, to which we have before alluded.

This speaking, then, from a mere impulse of imitation—the speech of parrots and of children learning to talk—is consequently an *acoustic reflex.* The stimulus to the motor-centre of sound originates here in the acoustic centre, which receives the words as an orderly succession of sounds and transfers them to the former.

The difficult question now arises: *Does this acoustic transfer of sounds take place behind the cerebrum or in the cerebrum?* Wundt[1] is of the opinion that the auditory nerve is excluded from the territory of the true reflexes; that it brings about the numerous incitements to motion which it produces only through the cerebrum.[2] If this were correct, the imitation of sounds would be always effected through the brain.

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[1] Grundzüge der physiologischen Psychologie. Leipzig, 1874. S. 182.

[2] This is by no means quite correct. *Wundt* himself describes the acoustic reflexes of the tensor tympani (p. 496. Note 2).

This is not the case, at all events, with the senses of sight and of touch. Not only do impressions on the optic and tactile senses provoke numerous simple reflexes, but a number of complicated movements, such as those of running, jumping, flying, and swimming, are executed by animals after being deprived of their cerebrum. And it is not alone those animals which bring these powers into the world with them, but also those which have only acquired them after birth. They even suit these movements to impressions received through the eye in a way which may be taken as involving a mental exertion and judgment. Frogs deprived of their brains will spring past an obstacle when impelled to jump by some painful stimulus (Goltz). Birds and mammals, with all the brain removed except the corpora quadrigemina, will follow the movements of a lighted candle with the head (Longet [1]).

Now, is there really any difference between the acoustic reflex and that of sight and of touch? Is it always cerebral, or can it, like the latter, be brought about through the basal ganglia below the crura cerebri?

In an article by Maudsley [2] on the physiology and pathology of the mind, an experiment of Vulpian [3] is alluded to, which seems to point to the existence of an acoustic sensorium in the posterior part of the cerebrum. "The rat," says Maudsley, "perhaps from being hunted generation after generation, is a very shy animal, very suspicious and distrustful, flying before the slightest unusual noise. If such an animal be deprived of the hemispheres of its brain, together with the corpora striata and optic thalami, it remains quiet. But if a low sound, such as cats make, be now produced, the animal will make a spring, and repeat this as often as the sound is reproduced."

In contrast, however, to this result, which is vouched for by a celebrated observer, there are a series of experiments by Danilewsky,[1] which seem to make it doubtful whether, in the case of sound-reflexes, the basal sound-centre can be excited from the hearing organs in any other way than through the lobes of the brain. In the formation of articulate sounds, the respiratory centre must be also excited in a certain definite manner. If in animals deprived of their brain we are unable to excite this centre through the ear, sound-imitation can only be brought about through the brain. Now, Danilewsky found that when, on cats and young dogs narcotized with morphia, he applied a weak electric stimulus to the gray substance of the supra-Sylvian convolution (Owen), corresponding to Hitzig's facial nerve-centre or the posterior part of the corpus striatum, the breathing became slow, with a preliminary deeper inspiration. The same effect was produced by shouting in the ear. But, after removal of the lobes of the brain, this reflex action from the acoustic nerve disappeared. Such a mode of breathing corresponds to that which speech requires. An electrical stimulation of the deeper portions of the brain, on the other hand, caused more rapid breathing, which we know is incompatible with speaking.

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[1] Anatomie u. Physiol. d. Nervensystems. Uebers. v. *Hein*. I. S. 385.

[2] Physiology and Pathology of the Mind.

[3] Leçons sur la physiologie du système nerveux, etc. 1866. p. 548. On referring to the original, I find that the author, on the strength of this experiment, regards the pons as the excitation centre for emotional movements, and also as the centre in which the stimuli of the auditory nerves are transformed into sensations.

From this experiment of Danilewsky it would appear that acoustic sound-reflexes can only come to pass through the medium of the cerebrum proper. If this be correct, the following conclusion is forced upon us: as soon as the child imitates sounds, the training of the cerebrum and its cortex for the objects of speech, through the medium of reflex mechanisms, commences. The moulding and shaping of the primal sounds of the basal centre into the customary sounds of conventional language and into those modifications of syllables and words which the rules of speech require, have their origin in the brain proper. From the latter the paths to the basal centre and to the muscles of the organs of speech are traced out and levelled; and at different ganglionic stations through which the stimulus must pass, the cell-connections with the basal sound-centre are set up, through which alone the various vocal sounds are fully represented and the formation of syllables rendered possible. It requires, as a rule, several years before all the sounds can be produced, and many persons are unable their whole life long to complete the whole task.

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[1] Pflüger's Archiv f. d. ges. Physiol. Bd. XI. 1875. S. 128.

After the destruction of voluntary speech, the *power of repeating words spoken aloud by others* often remains to those aphasic individuals not ataxic, although unable of their own free will to utter these words. They are incapable of bringing the word-pictures to the recollection by conceptions, but articulation has not suffered. By the utterance aloud of others, the acoustic pictures of the words which can no longer be produced from within are presented to them from without, and so the reflex start to the corresponding movements of sound is rendered possible. The same occurs in the echo speech of feeble-minded persons, and with the same impulsive desire which leads the child to repeat words, whether understood or not.

In our own experience, aphasic persons are much more successful in the repetition of words spoken aloud by others when they fix their eyes carefully upon the mouth of the speaker. The optic picture of the sound movements affords a kind of support to the acoustic. Thus we see that the same means which assist deaf-mutes in the acquisition of phonetic language are of service in the case of those afflicted with aphasia.

There could be but one clinical observation which would support the very improbable theory that imitative reflex speech is solely accomplished by a direct communication between the auditory nerve and the motor nerves of speech in the basal centre of sound. After complete destruction of the hemispheric voluntary tracts, say of both capsulæ internæ, individuals so affected ought still to repeat words spoken by another; but, until such a proof is forthcoming, let us suppose that *imitation is invariably a function of the cerebrum.* Clinical experience of cortical disturbances of speech of an aphasic character is plainly in support of this view. Imitative reflex speech is only found in amnesic aphasia, in which simply the acoustic word-picture has been lost, but not in ataxic, in which the word as a motor sound-compound has vanished.

CHAPTER XVII.

Interjectional or Emotional Speech.—Its Preservation after the Loss of Voluntary Speech.—Explanation thereof.—Theories of Hughlings Jackson and Jaccoud.

Both among animals and men the emotions lead to numerous expressions, whose *general principles* have been investigated by Darwin[1] in his own peculiarly thoughtful way. He has shown that here, too, it is only by the discovery of the general laws of development of the organic world that we are able to fully comprehend the question, although it may for all that still be impossible to ascertain with certainty the cause of most of the forms of expression of the emotions.

Many human *emotional gestures* which are common to all races are to be found also among the lower animals, while others are peculiar to man. For instance, among the first may be named the trembling of fear and rage, the writhing of the body and shrieking of mental agony, which have a parallel in the allied reflex expressions of bodily pain. Again, there are the skipping and leaping of joy and joyous expectation, the showing of the teeth in rage and defiance, which have become refined off into a display simply of the side teeth among many men, as a sign of defiant scorn. Even the laugh of man has its analogue in the chattering of some apes. "The habit of weeping, however, must have been acquired at or after that period at which man branched off from the common progenitors of the genus homo and the non-weeping anthropomorphs" (Darwin).

A cardinal difference, however, between man and the animals, lies in *the power of expression of the emotions by articulate sounds*, which is only enjoyed by the former. Animals, also, employ unarticulated melodious and unmelodious sounds for the purpose of calling, warning, or enticing its kind, and especially the male to charm or arouse the female.[2] Darwin regards this as the primal custom and the earliest stage of development of the voice. Many animals have only acquired the habit of giving vent to sounds before unnatural to them, since they have been domesticated. Thus it is with our house dogs, and even with tamed jackals, which have learned to bark, a sound proper to no species of the genus, with the exception of the canis latrans of North America, which is said to bark. Some races of tamed pigeons, too, have learned to coo in a new and peculiar manner.

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[1] The Expression of the Emotions by Animals and Man. 1872.

[2] Even apes utter in anger sounds much resembling interjections. A young female orang made jealous by the attention of her keeper to another ape, showed her teeth slightly, and uttered a sulky noise like tisch-schist, turning her back on him at the same time (Darwin).

The fact mentioned above, that a *musical feeling* awakes much earlier in children than sense for words, is probably connected with the early existence of musical feeling in the animal world. Not only is it present in birds, but also in mammals. There is an ape, for instance, one of the gibbons, which produces exactly one octave of musical sounds, singing the scale in the same tones upward and downward; so that it may be said of this ape "that it alone of all the mammals can sing" (Owen). From an observance of this fact, and by the analogy in other animals, Darwin has been led to the following conclusion: "that the progenitors of the human race probably employed musical sounds before they had acquired the power of articulate speech, and that in consequence of this, the voice, when employed in any violent emotional excitement, always tends to assume a musical character, on the principle of association." Certain it is that the loudness, resonance, timbre, height and intervals of the voice are determined by the influence of the different excitements of the mind, as has been shown by the investigations of Herbert Spencer,[1] confirmed by Darwin's observations.

It is impossible to discover the primary causes upon which depend our natural emotional utterances found among all races in allied forms. The sounds expressive of contempt and abhorrence, however, such as "puh," "pish," may be explained by the fact that they correspond with the movements of the mouth and nose which we employ in repelling disgusting or repulsive odors and tastes. Later every unpleasant impression, every repugnant conception, may be turned off with this movement and interjection which has become established by habit.

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[1] Essays, Scientific, Political, and Speculative, 1858. The Origin and Function of Music. p. 359.

Now, if in the case of the primal sounds, which have their cause in emotions of enjoyment, we are led by Goltz's "croaking experiment," to seek the *source of excitation of the centre of sound by feelings* in the cerebrum, how much more so in the case of those interjectional utterances and sound-compounds expressive of emotions of joy, wonder, abhorrence, etc., which conventional language contains, and which have in many cases been borrowed from the language of intellectual intercourse. But the feeling to which the emotions may be referred is no special property of the mind seated in a special province of the brain. It is nothing else than the mind viewed from this special point of view: in what strength and form of expression of feeling does the inner man react towards impressions when it estimates the latter in reference to the furtherance of its own well-being? The child and the savage react with coarse, sensuous feelings, and form estimates according to raw, sensuous experiences; culture fines off the feelings and mode of estimating things to an ideal elevation or refined commonness.

Emotional stimuli are far more powerful than those which are generated by imitation or accompany thought. They spread themselves, consequently, as a rule, not only over the higher and lower cerebral centres of motion, but also over the spinal and even sympathetic ganglia of the viscera. To this strength of the stimuli is due the fact that the emotional speech of children and savages, in whom it is unrestrained, is always accompanied by lively and characteristic gestures.[1]

The original natural sounds are all combined with gestures—they are "*sound-gestures.*" In the imitation of sounds, on the contrary, the stimulus is more restricted to the mimic and sound reflex-centres, while the emotional stimuli spread themselves out over the widest areas of the nervous system, not only affecting the motor-centres for the limbs and trunk, but also those for the breast, and even for secretion, and even acting on the functions of the senses and intellectorium. The more we acquire the power of representing abstract conceptions by words, and of restraining the feelings by understanding and reason, the more does the stimulus in speaking become restricted to the motor territory of the proper apparatuses of speech. Finally, philosophical thought, soaring into the loftiest abstractions, is accomplished best in silence. Softly spoken word-pictures or written pictures, which only let loose very weak stimuli upon the motor-centres of speech, are the most effectual in causing the conception of thoughts and guaranteeing thorough deliberation. Wise men and deep thinkers are, we know from experience, very sparing of words, while children and shallow-pates babble out whatever occurs to them.

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[1] Northerners accompany their language with fewer gestures than do southerners. The Englishman concentrates almost all his expression of feeling into language, while the Italian will act a whole comedy in pure pantomine.

Now, since emotional stimuli are far stronger than those accompanying quiet thought, and extend themselves over far wider tracts of the nervous system, it is easy to conceive that *the capacity of producing words for the purposes of expressing thought, or only for their own sake, may be lost, while emotional speech, or the capacity of uttering interjectional words, very often continues to exist.* The latter manifests a certain independence of the former.

This fact, if not first noticed, was at least first brought into prominence by that able observer, Hughlings Jackson.[1] Many very interesting experiences bearing upon this point have been recorded both by him, and by Trousseau, Broca, Gairdner, Broadbent, and others. Persons become aphasic, *i. e.*, incapable of uttering a single word voluntarily, are shown to have given vent to interjections, not only short words such as the complaints, O! Ach! Auau! which are very common, or Yes! and No! which are often uttered as interjections, but even longer and often very difficult words, as for instance, "Shocking!" or imprecations, and even brief sentences.[2] Such aphasic persons have repeatedly been regarded in hospital, by their attendants or fellow patients, as malingerers, from the fact that after weeks of the most obstinate silence to all questioning and remarks made to them, they have suddenly in anger burst out with a long and violent oath. Jackson[1] and Sir Thos. Watson have even found that aphasic persons, unable to reply "No" to a question, have nevertheless been led to do so by suggestions designed to make them angry, *e. g.*, were they a hundred years old, or a thousand, or such like. One may be then, as Jackson puts it, *speechless, and yet not wordless.*

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[1] London Hospital Reports for 1864.

[2] During the revision of this chapter a laborer, quite unconscious and with all the symptoms (afterwards confirmed post-mortem) of acute meningitis, was brought into hospital. Within the first twenty-four hours he uttered not a single word, but in the

Jackson[2] locates the "*automatic*" power of uttering words in the right hemisphere of the brain, and the voluntary in the left. We shall see later on that this latter view may be granted to be correct in this sense that right-handed individuals practice the left hemisphere chiefly or exclusively for voluntary speech. But it seems improbable that we practice the language of emotion only in the right half of the brain, as Hughlings Jackson supposes, and for this reason, because emotional gestures are executed sometimes with the right hand, sometimes with the left, sometimes with both. In anger we at one moment unconsciously clench the right fist, at another both; in asseveration we raise the right hand, as also in taking a solemn oath, or place it upon our heart; an object of detestation we repel with that hand which lies on the side threatened with its unwished-for contact. This seems clearly to indicate that an emotional stimulus may move the members of the body from both hemispheres, and hence the thought suggests itself that an emotional outburst of sound-compounds also may be accomplished by both hemispheres, or, in other words, that from childhood on both halves of the brain are practiced for interjectional sounds and words, but only one for true intelligent speech. If this latter be deprived of its functions as regards language, the other still remains for emotional sounds.

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morning, as he was lifted from one bed into another in a comatose condition, he burst out with a long oath. In the evening he died without awaking from the coma.

[1] Brit. Med. Journal. 1871, Dec. 2. p. 641.

[2] At the same place. Conf. also Clinic. and Physiol. Researches on the Nervous System. Lancet, 1875.

Jaccoud[1] supposes automatic speech to be presided over by a basal centre of sound situated underneath the corpora quadrigemina, and which he tries to localize more precisely, as we shall see in the next chapter. According to him, we have here to deal with the products of reflex stimuli of this "spinal" motor and co-ordinating apparatus, which replies with sounds at one time to acoustic, at another to optic impressions, while through great exertions of the will also it becomes excited and betrays this condition by monosyllabic and always similar words. Now, if the words were really always monosyllabic, this theory would have something very attractive about it, especially as we know from Bell, Romberg, and others, that the voluntary functions of the facial and hypoglossal nerves may be lost while their reflex action remains intact, and conversely, that the reflex movements may cease while the voluntary are preserved.[2]

It is, to be sure, correct that many aphasic persons, even when unconscious, continue to repeat automatically over and over again simple articulated sounds expressive of pain, as, for instance, "Oh, me!" "Ah, dear!" until they become comatose.[3] Others, again, react to every question and every function with some senseless syllable, *e. g.*, "tan," as is related of several French patients. Such simple combinations of sound might be regarded as analogous to the spinal reflexes which issue from the lumbar cord in the form of simple but orderly movements (Freusberg). But the same man (Broca's case) who only answered with the syllable "tan," uttered from emotion a long oath (sacré nom de . . .). Now, was this oath articulated in the brain, and the short syllable "tan" in the basal centre of sound? The oath, at all events, is of a more reflex nature than the stereotyped answer "tan." And what are we to say of the senseless polysyllabic words which many aphasic patients employ as a stereotyped answer, or of the cases where they have command of two or three such words as "tschi, tschi," and "akoko," as occurred in a patient of Westphal?

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[1] Leçons de clinique médicale, etc. 1874. p. 65.

[2] More at length in Chap. XIX. and at the end of this chapter.

[3] It has recently been my lot to see a young man succumb to endocarditis complicated with pneumonia and pericarditis. A few days before death this led to an embolic cerebral apoplexy with right hemiplegia and aphasia, at first without any disturbance of the consciousness. This patient gave me to understand by the most expressive signs that he was about to follow his departed wife. He first pointed with the left hand to a portrait of the departed on the wall, and then up towards heaven. He had once before his illness confided to me that his wife had told him not long before her death that he would soon follow her. He was only able voluntarily to utter the vowel a. Besides this, and even after he had become unconscious and hardly reacted to a call from his mother, he kept continually repeating the complaining sound "Oje." It was only in the last hours of life, after he had become quite comatose, that he became completely silent. Autopsy refused.

Finally, we see gradual transitions to those cases one of which Broadbent[1] has related, in which a larger number of words give expression to the emotions forced out rapidly in the manner of interjections, while the power of voluntary speech is entirely destroyed, the intelligence remaining. It is impossible to sever these experiences forcibly from one another, and one and the same explanation must be found for all.

These facts are in accord with what Hughlings Jackson states, namely, that the *mimic movements of expression* of aphasic persons, such as smiling, laughing, and weeping, may sometimes persist as emotional reflexes, while they can no longer be produced voluntarily. Indeed, it may happen that these emotion-reflexes in aphasic patients are completely out of gear and burst forth on the least provocation and with the greatest violence, like the spinal reflexes in decapitated animals. Such cases are frequently met with in which, as when the patients are addressed by the physician, or after fruitless efforts to render themselves intelligible by words, they burst out into convulsive weeping. If they are made to laugh, this too is convulsive.

Again, the *power of expressing musical feeling is independent of phonetic speech.* Just as children are sometimes able, before they can speak, to sing a tune they have heard (Sigismund), so also is it with aphasic persons. Such a patient, unable to utter a single syllable besides the one "tan," sang "the Marseillaise" and "the Parisienne" quite well, but with no other text than this incessant "tan, tan, tan," etc., etc. (Behier.[2]) Indeed, it sometimes happens that, under the influence of musical stimulus, words will make their appearance which could not otherwise be uttered. Hughlings Jackson[1] tells of idiotic children not deaf-mutes, who were only in possession of a few words, but who could sing, and when singing could bring out fresh words which otherwise they had no power over.

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[1] Conf. Chap. VII. S. 602.

[2] *Falret*, Article "Aphasie," in the Dict. encycl. des Sciences méd. T. V. p. 620.

Fever, too, is said to have generated words in cases in which the will was totally unequal to the task, just as strong excitement of the brain by emotions is sometimes able to do it. Jackson[2] relates a case occurring in the experience of Langdon Down, in which a speechless idiot came out quite as a speaker during the delirium of fever! Aphasic persons, according to Brown-Séquard,[3] sometimes recover their speech during delirium.

In a case of complete abolition of the power to move voluntarily the tongue and the muscles supplied by both facials, Romberg[4] noticed that the ability to close the lids and to swallow, through reflex action, still remained. Phonetic speech was reduced to the mere utterance of the sound "ang" or "ong"; but when, in the course of the reading or of the conversation, the patient happened to laugh or smile, it was observed that all the facial muscles performed their functions correctly. But even from this experience it would not be safe to draw the conclusion that in this case the emotional excitement proceeded directly from a centre governing the motions of laughing, and situated at the base of the brain, beneath the cerebral hemispheres. Opposed to this assumption is the statement of Romberg, that the act of laughing is produced by what is spoken or read in the person's hearing—that is, by his understanding what is spoken or read. According to this view, the excitement would unquestionably proceed directly from the cerebral hemispheres. The power to laugh is also known to be preserved in bulbar paralysis, even after articulate speech is no longer possible. The mechanism at the base of the brain, which governs the act of laughing, is evidently less interfered with by processes which affect the facial nerve, than is the mechanism that controls speech; and the emotional path along which the necessary stimulus ordinarily travels is also capable of bearing greater disturbances than can that over which the stimulus of the will is transmitted.

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[1] Lancet. 1861, Sept. 23.

[2] Ibidem.

[3] London Medical Record. June, 1874.

[4] Loc. citat. p. 786.

CHAPTER XVIII.

The Phonic Centre below the Corpora Quadrigemina, or Basal Phonic Centre.—Its Share in the Formation of Articulate Sounds.—Theories of Cruveilhier, Leyden, Schroeder van der Kolk, and Jaccoud.—Peculiarities of Structure which seem to make the Medulla Oblongata a Suitable Locality for the Co-ordination of Phonic Movements.—Clinical Facts which render the Implication of the Medulla and Pons in Articulate Utterance a Certainty.—Disorders of Articulation in Progressive Bulbar Paralysis.—Significance of the Bulbar Nuclei.—Bulbo-nuclear Stammering.—Disorders of Articulate Speech consequent on various other Lesions of the Medulla Oblongata and Pons, likewise on Insular Sclerosis of the Brain and Spinal Cord.—Basal Kinesodic Dysarthries, Intracerebral and Extracerebral.—Bradylalia and "Scanning" Utterance.—Relation of the Mechanism of Phonation to the Synthesis of Syllables and Words.—Stammering, Stuttering, Blundering Enunciation of Syllables, Atactic and Amnesic Aphasia.—The Cerebellum in its Relation to Articulate Utterance.

Animals which express their feelings by screaming, crowing, croaking, and similar *non-articulate* sounds, may still be induced to utter these sounds after their brain, up to and including the *corpora quadrigemina*, has been removed. Anencephalous monsters and infants craniotomized during delivery [1] are likewise capable of emitting cries and squeaks. *The reflex-centre for such non-articulate sounds must therefore be situated below the corpora quadrigemina, its lower limit in the cord coinciding with that of the respiratory centre.* This reflex-centre—whose integrity is undoubtedly essential for the production of *articulate* as well as of non-articulate sounds—we shall term the *basal phonic centre.*

In affirming that articulate sounds cannot be produced without this basal centre, we do not affirm that it is itself the centre for articulate utterance, understanding by this the workshop wherein the formation of literal sounds actually takes place; the letter, as an acoustic image, being transformed in accordance with the phonetic laws of speech into co-ordinate movements of the respiratory organs, the larynx, and the mouth (which contributes to the formation of vowel-sounds and consonants). Before making any such assertion it would be necessary to prove that this delicate elaboration of crude sounds into the phonetic instruments of the intelligence is capable of being effected within the narrow limits of that basal region.

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[1] When I was a student I had an opportunity of dissecting a well-developed anencephalous fœtus, born at full time, which was said by the midwife to have squeaked like a mouse several times after its birth.

True, writers of authority have confidently localized articulate phonation in the pons and medulla oblongata, with or without the addition of the cerebellum.

As long ago as 1834, Cruveilhier[1] arrived at the conclusion, based on several instances of atrophy of the medulla, either by itself or together with the pons, in which the power of articulation was lost, that the centre for articulate phonation must be seated in those parts.

Leyden[2] expressed the same opinion in 1867.

Schroeder van der Kolk[3] ascribed the power of regulating the contractions of the muscles concerned in speech to the *olivary bodies*. He believed himself to have discovered that they are connected with each other and with the facial and hypoglossal nuclei by commissural fibres. This hypothesis must now be definitely abandoned. Neither Deiters nor Meynert has been able to find any such commissural fibres, and Clarke has shown that the evidence furnished by comparative anatomy is dead against the theory. The parrot and other animals capable of mimicking articulate speech possess no olivary bodies, or possess them only in a very rudimentary form; while the seal (Seehund), whose reputation for eloquence still remains to be made, has them of enormous size.

Jaccoud[4] at one time followed Schroeder in placing the centre for articulation in the olivary bodies. Recently, however,[1] he has transferred the function of co-ordinating the movements of phonation to the "*système commissural cérébello-bulbaire, i. e.*, the bulbar commissure of the olivary bodies, together with the commissure of the middle crura of the cerebellum, both in the intra-cerebellar and in the more central—intra-pontine—portion of its course. As regards the olivary commissure, its importance in relation to articulate speech is much weakened by the researches of Clarke (vide supra). The significance of the cerebellum and its pontine commissure is based by Jaccoud on a case accurately observed by Meynert. But this case is not altogether conclusive.[2]

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[1] Arch. génér. de médecine. Juillet, 1834. IV. 416.

[2] Berlin klin. Wochenschrift. 1867. p. 78.

[3] Bau und Funktionen der Medulla spin. und oblongata. pp. 161, 165.

[4] Gazette hebdomadaire. 1864, Juillet 22.

It must be admitted that *various features in the anatomy of the medulla oblongata appear to render it a suitable locality for the co-ordination of the movements concerned in phonation.* First, it contains the two great vago-accessory nuclei—the sensori-motor nuclei for the respiratory and vocal movements which are auxiliary to speech. Secondly, the motor nuclei of all the nerves which take part in the muscular movements of articulation lie serially disposed within a narrow space in the medulla oblongata. Thirdly, the corresponding nuclei of those nerves are symmetrically arranged on either side of the raphé, in such close proximity to each other that, by means of commissural fibres (whose existence has been demonstrated, at any rate for the hypoglossal nuclei), a simultaneous contraction of the homonymous muscles of the tongue, lips, palate, etc., may readily be effected, even when the impulse is propagated from one side only. Fourthly, the medulla contains the nuclei of the *portio mollis* and the fifth nerve—the sensory nuclei of hearing and touch. Fifthly, we may fairly assume that these sensory and motor nerve-nuclei, which are probably concerned in the reflex act of phonation, are connected with one another by numerous fibres, although, as might be anticipated from the difficulty of the investigation, only a few such connections have hitherto been demonstrated. Meynert,[1] more particularly, asserts that the facial, vago-accessory, and hypoglossal nuclei are connected with the fasciculi arcuati and acoustic nuclei by a system of intercalated nerve-corpuscles, abundantly furnished with processes, and surrounding the nuclei on every side. Finally, there is reason to believe that acoustic fibres penetrate into the cerebellum likewise (Clarke); and this leads Meynert[2] to speculate that rhythmical auditory impressions may, through the cerebellum, determine the rhythm of movements, and thus perhaps of uttered sounds likewise.

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[1] Leçons de clinique médic., etc. 1874. p. 73.

[2] In a case of sclerotic shrinking of the pons and cerebellum, attended with muttering speech, *Meynert* found the roots and nuclei of the hypoglossal nerves and the conducting fibres of the pons uninjured, while its transverse fibres had undergone degeneration. But might not the cicatricial shrinking of the pons have impaired the conductivity of the longitudinal fibres, thereby giving rise to the defect of speech? (Wien. med. Jahrbüch. 1864. p. 4.)

We possess, moreover, valuable evidence on the clinical side, which puts the importance of the basal parts of the brain in reference to articulate phonation beyond all doubt, and proves, more particularly, that this function depends on the integrity of motor nuclei in the medulla oblongata.

The last-named fact has been securely established by the more precise investigation of the structural lesions in progressive bulbar paralysis (Leyden, Charcot, Duchenne, Joffroy, Hun, R. Maier), and in those degenerations of the gray medullary columns of the spinal cord which ascend to the medulla oblongata (Clarke). *The integrity of literal phonation is bound up with the integrity of the motor nuclei in the medulla oblongata*, more especially of the hypoglossal, but also of the vago-accessory and facial nuclei.[3] In two cases recorded by Charcot and Duchenne-Joffroy, the only abnormality that could be detected in the medulla oblongata was degenerative atrophy of the corpuscular elements of the motor nuclei.[4] In the other cases, sclerotic changes were likewise met with in the anterior pyramids and other motor tracts. On the other hand, the sensory nuclei, more particularly those of the auditory, fifth, and glosso-pharyngeal nerves, were hardly ever, the restiform bodies never, found affected.

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[1] Archiv für Psych. Bd. IV. p. 419.

[2] *Meynert*, Wien. med. Jahrb. a. a. O.; and *Wundt*, Grundzüge der physiolog. Psycholog. 1874. p. 220.

[3] See my lecture on Progressive Bulbar Paralysis, in Volkmann's Sammlung klin. Vorträge, No. 54.

[4] I am quite aware of the difficulty there often is in drawing the line between physiological pigmentation of the ganglion-cells and that granular degeneration which leads to their becoming atrophied and destroyed. This, however, in no way alters the

As in the course of these degenerative changes one ganglion-cell after another is slowly destroyed in the bulbar nuclei, we perceive consonants and vowels successively crumbling away, as it were, from the patient's speech, while his intellectual powers may be perfectly retained. His words grow more and more indistinct and mutilated, his stammering passes into an unintelligible muttering, until at last he is only able to emit grunting noises, and perhaps not even these. So long as his hands and arms escape paralysis, he continues to communicate his feelings and ideas by writing. The disorder of speech is invariably limited (when there is no cerebral complication) to the power of forming articulate sounds; the stammering may pass into complete loss of speech, but it is never associated with a blundering enunciation of syllables (Sylbenstolpern), or with aphasia. *The degree in which the formation of syllables and words is interfered with is always proportionate to the disorder of literal phonation;* the architecture, so to speak, of the words is not impaired; they continue to be correctly put together, though the materials—the elementary sounds of which they are made up—may be inadequate.

The order in which the elementary sounds of the alphabet are lost in this disease is not invariably the same. It depends on the order in which the groups of muscles concerned in the articulation — those of the lips, the tongue, the palate — become paralyzed. Should the lips be the first to fail, *o* and *u* (oo) are the first sounds to be obliterated; *i* (ee) and *e* (eh) soon follow them. *A* (ah) lasts as long as phonation is possible at all. When the tongue loses the power of rising from the floor of the mouth before the lips become affected, *i* (ee) is the first sound to go. The failure of the tongue robs the patient first of *r* and *sh*, then

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fact that the atrophy and destruction of these cells entails the annihilation of their functions.

of *s*, *l*, *k*, *g* (hard), *t*, lastly of *d* and *n*. Palsy of the lips interferes with the pronunciation, first, of *p* and *f*, later, of *b*, *m*, and *v*. Paralysis of the soft palate gives the voice a nasal twang. When sufficiently advanced to permit a considerable escape of air through the nose, it hinders the pronunciation of the labials *b* and *p*, which come to resemble *m*, *w*, or *f*, because, as Duchenne was the first to point out, the current of air directed through the mouth is no longer powerful enough to burst through the tense and compressed lips. Should this firm closure of the tense lips still continue practicable, the patient will be able to utter *b* and *p* when his nose is pinched, and this shows Duchenne's explanation of the facts to be correct. We have here our first example of a *paralalia* or *pararthria literalis*, whose mechanism is, moreover, fully made out.

The importance of the motor-nuclei of the medulla for the production of articulate sounds is thus securely established. Their destruction is followed by paralysis and wasting of the muscles concerned in speech, and ultimately, therefore, by loss of speech.

Duchenne denied that paralysis from disease of the bulbar nuclei was followed by muscular atrophy ; but his denial was only based on the fact that the atrophy is not always to be recognized during life. The wasted muscles may be so completely replaced by fat that the size of the tongue is not diminished. *Paralysis and atrophy of the tongue, together with fibrillary twitchings of its muscles and associated disorders of deglutition are invariably associated with bulbo-nuclear stammering — Anarthria literalis bulbo-nuclearis — and must accordingly be viewed as essential diagnostic marks of the disease.* The reflex excitability and electro-contractility of the palsied muscles appears to depend on the degree of nervous and muscular degeneration (Rosenthal[1]). As a general rule, the paralysis is limited to the respiratory division of the facial nerve (supplying the mouth and nose), while the orbicularis palpebrarum retains its functional activity. The fact is a striking one, and has not hitherto been explained.

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[1] Klinik der Nervenkrankheiten. Wien, 1875. p. 252.

It is probable that the motor-nuclei of the medulla, or their constituent nerve-cells, exert an important influence upon the muscles of articulation, an influence similar to that exerted by the large cells of the gray matter of the anterior columns of the spinal cord on the muscles of the trunk and limbs. The nutrition and irritability of the muscles in connection with the spinal cord is closely bound up with the integrity of these cells, which is therefore an indispensable condition of all movement, whether reflex or voluntary. And since, even in the higher mammals, the spinal cord is capable of co-ordinating the simpler movements after its separation from the brain (Freusberg), we may by analogy ascribe a similar power to the medulla oblongata. Moreover, we learn from Exner's experiments,[1] that a stimulus setting out from the brain undergoes retardation in the ganglia of the caudex cerebri (Stammganglien); that it subsequently traverses the spinal cord with great rapidity; that, lastly, it is once more delayed, and at the same time intensified, before entering the nerve-roots. There is nothing against the view that the primitive sounds emitted by the infant at birth are movements of so simple an order as to find the necessary mechanism for their co-ordination adequately supplied by the medulla oblongata alone. On the other hand, it is in the highest degree doubtful whether this bulbar mechanism is of itself sufficient for the production of all the elaborate sounds required by language—sounds which have to be acquired for the most part with much expenditure of time and pains, and resembling in this respect the art of standing erect, of walking, of handling tools, etc. What is true of these movements, which cannot be accomplished without the aid of cerebral ganglia, more especially of the corpora striata, must be truer still of the constituent elements of language, whose elaboration is yet more closely bound up with the intelligence, and which are from the first combined with syllables and words, whose value and meaning are determined, not indeed exclusively, but principally by the arrangement and collocation of their constituent sounds.

Dysarthric impediments of speech, from stammering to complete inability to utter sounds, may be caused, not only by the gradual degenerative processes underlying progressive bulbar paralysis, but also by various *acute and chronic morbid changes in the medulla oblongata*, when they involve the motor, especially the hypoglossal, nuclei. To this category belong hemorrhagic extravasations, circumscribed necrotic and inflammatory changes, abscesses, tumors, islets of sclerosis, etc. So, too, injury or disease of the skull and its periosteum, pressure of cerebellar tumors, extension of inflammatory and degenerative processes from the pons or from the spinal cord to the region of the motor nuclei, may damage the medulla to such an extent as to interfere with articulate phonation.[1]

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[1] *Pflueger's* Archiv. VIII. p. 526.

But even when the integrity of the bulbar nuclei is unaffected, disease of the pons alone, or of the pons in conjunction with the medulla, may hinder articulate utterance by interfering with the transmission of impulses from the cerebrum to the nuclei and the muscular mechanism of speech. Under such circumstances articulate phonation becomes imperfect, either in *time* or in *form*. In the former case, vowels and consonants are correctly and distinctly uttered, but their enunciation—whether singly or in combination—is too slow, or interrupted by pauses between the syllables, or uneven in rhythm. The channels leading to the muscles of articulation are still all of them open, but they are obviously less smooth than usual, or perhaps less numerous—single fibres or bundles of fibres having been destroyed, and the impulses being obliged to follow a circuitous route. In cases of the second class the transmission of impulses is more seriously hindered, and can no longer be effected even along collateral channels; the enunciation of both vowels and consonants is stammering and imperfect, and the faculty of articulate utterance may at length be wholly annihilated. Such dysarthric impediments of speech may be contrasted with the "basal nuclear" group as *basal kinesodic dysarthries* ; they may be further qualified as *central* or *intra-cerebral*, to distinguish them from those *peripheric* or *extra-cerebral, though still intra-cranial, dysarthries*, which are due to lesions affecting the motor nerves of speech after they have issued from the medulla oblongata.

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[1] Cf. *P. Déchery*, Quelques formes d'atrophie et de paralysie glosso-laryngée d'origine bulbaire. Thèse. Paris, 1870.—*Rosenthal*, Allg. Wien. med. Ztung. 1867. 15 and 16.—*The same*, Klinik der Nervenkrankheiten. 1875. pp. 195-201, and 237-258.—*Leyden*, a. a. O. and in his klinik der Rückenmarkskrankheiten. 1875. Bd. II. p. 157 (Myelomalacie des Bulbus) and p. 65 (Blutextravasat in der med. oblong., der unteren Hälfte des Pons, und dem linken mittleren Kleinhirnschenkel).—*Wilks*, Guy's Hosp. Rep. 1870. II. p. 67.—*Bälz*, Arch. d. Heilk. 1872. p. 192.—*Jackson*, Lancet, 1872. No. 30.—*Eichhorst*, Charité-Annalen. Berlin, 1876. p. 206.—*Friedreich* observed stammering come on during the extension of tabetic degeneration of the posterior columns to the med. oblongata, Virchow's Archiv. XXVI. pp. 391 and 433.

It is probable that these "intra-cerebral basal kinesodic dysarthries" are not attended, like those of "nuclear" and "extra-cerebral" origin, by atrophy of the muscles of speech. Moreover, in marked contrast with what occurs in nuclear stammering, the tongue may occasionally be observed in these cases to retain the power of executing every kind of movement at will, both rapidly and correctly ; it can be protruded far beyond the teeth, raised with seeming vigor towards the palate, moved sideways and backward ; chewing and swallowing may be satisfactorily accomplished, even after speech has grown very indistinct. These facts may be explained in one of two ways : we may assume that the transmission of phonic impulses is more hindered than that of impulses for other movements, the paths along which the former travel being specially damaged ; or—and this is the more likely theory—we may suppose the transmission of all impulses to be equally impeded, and all the voluntary movements of the tongue to be hampered alike ; the paresis of those ancillary to speech being more obvious, however, than that of the rest, which can only be detected by the employment of special methods. We may notice, for example, that the patient is unable to make a clacking noise with his tongue as forcibly and readily as he used to do ; that he soon gets tired of chewing and swallowing. The paresis of the lips, too, is often betrayed only by the diminished loudness of the patient's whistling. As regards electrical methods of diagnosis, Rosenthal affirms that, in paralysis due to tumors in the pons, faradic excitability of the facial muscles and nerve-twigs may be abolished, galvano-muscular contractility exalted, galvanic irritability of the branches of the portio dura diminished or altogether destroyed, just as in paralysis of the facial nerve from extra-cerebral causes. Whether the same be true of the tongue likewise, we do not know.

That articulation is *not invariably* interfered with by circumscribed lesions of the medulla oblongata and pons, may readily be explained if we reflect that they are neither of them simple, but composite organs, made up of a great variety of functionally distinct centres and conducting fibres. The consequences of a lesion always depend on its precise extent of situation. At the present time we know only that for articulate utterance the motor nuclei of the nerves supplying the organs of speech are of prime importance, the olivary bodies of no importance at all. Concerning the part played by the various regions of the pons and medulla individually in relation to this function, we know nothing. Whether the commissural fibres of the middle cerebellar peduncles, in the central portion of their course, where they traverse the lower part of the pons, really possess the significance—as regards articulation—ascribed to them by Meynert and Jaccoud, remains to be proved by evidence of a more conclusive kind than has been hitherto adduced.[1] It is to the precise topographical determination of small, sharply circumscribed hemorrhagic cicatrices and patches of necrotic softening in the substance of the pons and medulla oblongata, that we must look for information concerning the function of the individual basal areas in regard to the faculty of speech. It will be as necessary to give an accurate description of those lesions which are *not* attended by any disorder of speech during life, as of those which *are*.

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[1] *Darolles* (Progrès médic. 1875, p. 629) describes a sharply-defined patch of thrombotic softening, as big as a small hazel-nut, which was situated in the right upper half of the pons; it did not transgress the median line, and stopped short of the pedunculus and the point of origin of the middle crus of the cerebellum. This lesion had given rise (the patient was a woman of thirty-six) to complete right-sided (not alternate) hemiplegia; the tongue was nearly motionless, articulation very imperfect, intelligence unimpaired. The basilar artery was plugged by a thrombus. In the year 1873, I myself came across a patch of softening situated in the right upper half of the pons of a bricklayer, aged fifty-nine. It was as big as a cherry-stone, marbled with red and gray, and consisted (under the microscope) of a mass of granule-corpuscles amid a fibrillar reticulum. It did not transgress the middle line, but penetrated through the inferior and middle zonular layers (Gürtelschichte), together with the longitudinal fibres that lie between them. No plugs were found in the arteries. The patient had had a slight apoplectic stroke in August, 1873, after which his left side was paralyzed for three days. About the twelfth of December he fell ill again. Along with fever of a markedly intermittent type (evening temperature from 38° [100.5° F.] to 41.5° C. [106.9° F.], morning temperature from 36.2° [97.2° F.] to 39° C. [102.2° F.]), he was seized with complete paralysis of the left side, including the cheek; sensation was now and then diminished in the palsied limbs; the left pupil was permanently and excessively contracted; the tongue freely movable, with occasional deviation towards the left. He was often delirious, but answered questions for the most part correctly; no fault

The study of the disease known as insular (disseminated) sclerosis of the brain and spinal cord is of considerable interest in reference to the centres and channels subservient to articulate phonation.

The disorders of speech met with in this disease are almost invariably dysarthries. The nature of the disorder depends wholly on the seat of the sclerotic change. In the purely spinal form of the disease, articulation—to judge from a single case—does not appear to suffer.[1] Should the changes be confined to the medulla spinalis and oblongata (bulbo-spinal form), everything will depend on their distribution in the substance of the latter.[2] Lastly, in the cerebro-spinal form of the disease, disorders of speech, or at any rate of the voice, seem always to exist. The disorders of speech always involve the faculty of articulate utterance, hardly ever the actual structure of words (atactic aphasia), or of sentences; for the cortex cerebri is seldom affected by the morbid change, and then, as a rule, only to a trifling extent. The cortex cerebelli shares the almost absolute immunity of the cortex cerebri. The islets of sclerosis are

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was noticed in his articulation. He died on December 25th.—These two observations are contradictory, and the contradiction does not at present admit of reconciliation.

[1] *Pennock's* cases, recorded by *Morris*. Cf. *Bourneville* and *Guérard*, De la sclérose en plaques disséminées. Paris, 1869. p. 76.

[2] In *Vulpian's* case (*Bourneville*, l. c., p. 64) no mention is made of any disorder of speech; the sclerosis was limited to small islets in the restiform bodies, the olivary bodies, and the floor of the fourth ventricle.—On the other hand, in *Ebstein's* case (Deutsch. Archiv für klin. Medic. Bd. X. p. 599), the sclerosis gave rise to monotonous, but not scanning utterance, with indistinct formation of literal sounds, *i. e.*, with stammering. In this case the insular deposits did more than merely interrupt the fibres of the hypoglossal nerves in the intra-medullary portion of their course; they likewise penetrated into one of the hypoglossal nuclei, destroying its cells.

chiefly disseminated through the central region of the greater brain, the corpus semiovale with the gray matter of the central cavities (Höhlengrau), the corpora striata and optic thalami; they frequently increase in number as we proceed downward, in the pons and medulla oblongata. Broadly speaking, the motor centres and conducting fibres are more affected by the sclerosis than those concerned in sensation; still, I cannot agree with Buchwald,[1] in terming the entire disease a "sclerosis of the motor centres," and contrasting it with tabes dorsualis.

Leube[2] has pointed out that *symptoms of glosso-pharyngeal paralysis* constitute a part—though usually but a small part—of the clinical phenomena presented by insular sclerosis of the brain and cord. Others[3] have confirmed this statement. The fact is not surprising, inasmuch as the sclerosis may lead to atrophy and complete destruction of the cells in the bulbar nuclei.[4] The patient begins by stammering, his utterance passing ultimately into an unintelligible muttering.

But there are also cases in which the faculty of articulation exhibits no such decided impairment. The power of modulation is lost; the voice is pitched in a monotone, and is occasionally nasal. Utterance is not indistinct; it is merely slow, notwithstanding an unusual expenditure of effort (*bradylalia*, or *bradyarthria*). Letters and syllables, though correctly formed, no longer succeed one another with their customary rapidity. If the syllables are separated from one another by marked pauses, we call the utterance "*scanning*." The patient continues to possess full command over his tongue, and his power of swallowing is not impaired. He suffers merely from difficulty and delay in the transmission of motor impulses to the muscles of the respiratory apparatus, of the larynx and tongue. The channels are no longer pervious; here and there, in the greater brain and mesencephalon, they are interrupted; more powerful discharges of nerve-force are required, and they are probably conveyed in part along collateral paths.[1] We must also attribute to disturbances of transmission such symptoms as those noticed in a case of Schuele's, where "individual syllables were shot out with explosive vehemence, while those immediately succeeding them were uttered in the hesitating, scanning fashion characteristic of the disease." This sort of ataxy depends on a false estimate of the amount of force required for articulation, due to altered resistances in the conducting fibres. Closely related to this phenomenon is another, which I had occasion to observe, for years together, in a woman suffering from insular sclerosis of the brain and cord (case published by Dr. Putzar). After her death we found very extensive sclerosis of the medullary layer of both hemispheres (just as in Schuele's case), besides a number of islets of disease elsewhere. Her speech continued quite intelligible to the last; articulate sounds, apart from slight hesitation and sibilation, were correctly formed; but her words, without being actually "scanned," were jerked out in a fragmentary sort of way, in a somewhat bleating tone of voice, and with decided effort. The hypoglossal nuclei and the intra-bulbar hypoglossal fibres were found intact, on microscopical examination of the hardened tissues.[2]

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[1] Deutsch. Arch. f klin. Med. Bd. X. p. 478.

[2] *Ibid.* Bd. VIII. p. 1.

[3] Conf. *Schüle*, *ibidem.* VIII. p. 223.

[4] As in *Ebstein's* case, referred to above; also in one recorded by *Joffroy*, Gaz. méd. de Paris. 1870. No. 23.

It is hardly necessary to state that utterance may be rendered stammering to unintelligibility, notwithstanding perfect integrity of the bulbar nuclei and basal conducting fibres, solely in consequence of *blocking of those channels in the hemispheres along which impulses are conveyed downward from the cortex cere-*

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[1] In a case of insular sclerosis of the cord, medulla oblongata, and pons, without implication of the cerebrum, which occurred in my wards, and has been published by Dr. *Engesser* (Deutsch. Archiv f. klin. Med. XVI. p. 556), the only fault detected in the patient's utterance was its slowness and slightly hesitating character. The hypoglossal nuclei were unaffected. The conducting channels were interrupted here and there in the pons and medulla oblongata. The cerebrum appeared to be in a perfectly normal condition.

[2] Speech is often accompanied by noisy (jauchzend) inspirations. *Leube* attributes this to paresis of the crico-arytænoidei postici, whose function it is to widen the opening of the glottis. *Hirsch* (Deutsche Klinik. 1870. Nos. 33 to 38) met with a case in which the patient's slow and indistinct utterance was repeatedly interrupted by laughter. This reminds me of a gentleman suffering from bulbar paralysis, who would burst into shouts of laughter on the smallest provocation.

bri. This is proved by a case of Jolly's,[1] in which the corpus callosum and the medullary substance of the cerebral hemispheres were very extensively indurated and of leathery toughness, while the great ganglia—the lenticular nuclei, corpora striata, and optic thalami—together with the gray matter of the cerebral peduncles and the bulbar nuclei, were altogether intact. Towards the close of her life the patient's stammering grew so extreme that she was only able to utter the words "yes" and "no" intelligibly. The symptoms came generally to resemble those of progressive bulbar paralysis, though the sclerosis was limited to the white medullary substance of the hemispheres. The sensorium remained unclouded to the end. It is worthy of note that, at a time when her consonants had become almost unintelligible, the patient was still able to utter all the vowel-sounds in a loud tone of voice, almost destitute of modulation. After she had ceased to be intelligible at all, she still, when she wanted to speak, emitted loud, clear sounds in a high-pitched monotone. If she tried to put out her tongue (which did not appear to be wasted), she failed in the attempt, the tongue rolling helplessly about in her mouth. These peculiarities, which have not hitherto been observed in progressive bulbar paralysis, may possibly serve as a clue to diagnosis in future.

I consider it a fact of great importance that in insular sclerosis, even when the islets of degeneration are of considerable size and abundantly disseminated through the medullary tract of the cerebrum and down into the cord, it is only the mechanism of articulate phonation and the rapidity of utterance that are impaired—never the co-ordination of syllables and words in obedience to the laws of spoken language, unless the cortical substance be simultaneously implicated in the degenerative process. We never meet with the blundering enunciation of syllables (Silbenstolpern) so common in general paralysis, or with true aphasia. The "scanning" utterance of syllables must not be confounded with blundering enunciation. In the former, articulate sounds are correctly combined to form syllables and words; the syllables are merely separated from one another by brief pauses. In the latter, both articulate sounds and syllables are misplaced and thrown into confusion. To borrow Westphal's admirable illustration, the general paralytic, in trying to say "artillery," calls it "artrillerary;" the patient with insular sclerosis, on the other hand, pronounces it "ar-til-le-ry."

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[1] Archiv f. Psych. Bd. III. 1872. p. 711.

Stuttering (Stottern), again, has not hitherto been observed in cases of insular sclerosis. But this is not a point on which I am inclined to lay much stress in connection with the localization of the functions concerned in speech; for it depends far more on the nature of the process (sclerosis) than upon its situation. Stuttering is not, like stammering, a paralytic defect of articulate phonation; it is a spasmodic hinderance to the enunciation of syllables, and more particularly to the vocalization of the necessary sounds. It requires for its production an exaggerated excitability—exaggerated to the point of generating spasm—in that part of the articulatory centre which is specially concerned in vocalization. This state of spasmodic excitability may be induced by influences propagated from the most diverse points of the nervous system; it may be caused by irritation of the bowel no less than by irritation of the spinal cord or brain. Hence, we must not jump too hastily to the conclusion that the seat of the lesion to which the stuttering is due is at the same time the seat of the faculty by which the vocalization of syllables is effected. There is, indeed, some ground for the opinion that morbid changes at the base of the brain, when giving rise to considerable irritation, may cause stuttering. Cases are on record of this symptom having been brought on by tumors and other lesions in this neighborhood. Unfortunately, stammering has not always been clearly distinguished from stuttering; the terms used formerly to be employed without due discrimination; hence, the older observations bearing on this point do not carry much weight.

The present is a suitable opportunity for entering somewhat more deeply into *the relations between the mechanism of articulate phonation* (Lautmechanik) *on the one hand*, and *the construction of syllables* (Silbenfügung) *on the other.*

The successful construction of syllables depends for its realization on the following conditions: First, the muscles of speech must be individually capable of adequate contraction. Secondly, the phonic-respiratory groups of muscles in the trunk and larynx must co-operate accurately with the articulatory muscles of the mouth. Thirdly, the groups of muscles which form consonants, and those which form vowels, must fulfil their respective offices. Fourthly, the attitude of the organs of speech in pronouncing consonants must be correctly joined to their attitude in pronouncing vowels, and the successive transition from one attitude to another must be suitably accomplished.

Stammering (Stammeln) consists in a failure of the muscles of speech as regards both their individual contractions and those combined contractions which are requisite for the enunciation of consonants and vowels. The obstacle to their independent or their co-ordinate contraction may be of a coarse, mechanical nature, situated in the jaws, teeth, palate, etc., or in the muscles themselves; or it may be peripheral, situated in the motor nerves of the tongue, palate, and face; or it may be central, due to some failure on the part of the motor centres to originate impulses of sufficient power to put the muscles of speech in action; or, the transmission of the impulses may be disturbed, or they may be diverted into wrong channels.—Broadly speaking, the imperfect utterance of syllables is due to some imperfection in the working of the external or internal mechanism of articulate phonation; *the stammering may be of external or of internal origin.*

It is clear that when stammering is produced by any of the above-named causes, the transit of the muscles from one attitude to another, and the fusion of consonants with vowels ("vocalization of consonants"), will likewise be interfered with; in other words, *the imperfect utterance of elementary articulate sounds will entail an imperfect utterance of syllables.*

But imperfect formation of syllables may also coexist with integrity of the mechanism of articulate phonation; it may exist without stammering, and altogether independently of it. Stuttering and the blundering enunciation of syllables (Silbenstolpern) are purely syllabic faults of speech, and must be rigorously distinguished from stammering.

Stuttering (Stottern) is due, not to any peripheric hinderance of a mechanical kind to the activity of the organs of speech, nor to any central failure of motor power, nor yet to any interruption of its transmission; it is due simply and solely to a spasmodic inability to execute that "vocalization of consonants" (union of consonants with vowels) which is requisite for the formation of syllables. The attempt to fuse a consonant with a subsequent vowel into a syllable, sometimes even the attempt to utter a syllable beginning with a vowel, brings on spasm of the muscles of speech—the spasm not being confined to the muscles actually engaged in the formation of particular letters, but spreading to the phonic and respiratory muscles likewise; nay, the central irritation occasionally radiates over many other muscular regions also. The speaker is left in the lurch, not by the centre for co-ordinating the movements of articulate phonation, but by that which presides over the "vocalization" of sounds with a view to the construction of syllables. How this "*vocalization-centre*" is connected with the centre for articulate phonation, we do not know.

In "*blundering enunciation of syllables*" (Silbenstolpern) the combination of articulate sounds into syllables is interfered with, not by any external mechanical obstacles to pronunciation, or to the connection of consonants with vowels; not by any central failure of motor energy for articulate phonation; not by any break in the transmission of impulses to the muscles of speech; not by any spasmodic hinderance to the "vocalization" of consonants; but by causes situated on a higher level. *The fault lies in the speaker's inability to co-ordinate articulate sounds into syllables and syllables into words, as organic units held together in conformity with the law of spoken language. The imperfection of the syllables results from the incorrect interpolation and collocation of articulate sounds—in themselves correctly formed and correctly joined with one another—in words, as the organic units of language, and as articulate motor-aggregates.* The completed word may be said, in a manner, to assimilate its constituent sounds, to incorporate into a single organic unity those elementary parts which derive their true significance only from their relative position and the stress laid on them. This organic whole—this compound organism of language—has its intimate structure loosened; its constituent sounds fall apart and get mixed up with one another; they fail to make their appearance at all, or they make it in the wrong place, too soon or too late, with a right or a wrong accent, never twice in the same order. Occasionally, moreover, letters which have nothing whatever to do with the word usurp the place of others that properly belong to it.

In *aphasia*, finally, the word as a whole is wanting, or it is uttered in some mutilated form, or one word is substituted for another. It is not merely the constituent letters of a word that drop out of it at random, or are misplaced, or wrongly accentuated, whereby the word is twisted out of shape; it is the word itself that drops out of our speech, or presents itself in a new and not seldom quite mutilated form. It is no longer the mere structure of the word that is shattered; the combination of words into sentences is at fault. The aphasia is termed "*atactic*" when the word, though still retained as a sensory image and as a symbol of thought, can no longer be enunciated as a motor combination of articulate sounds, though the sounds themselves may still continue to be correctly formed when occurring in some other word; but when the word no longer exists in consciousness, even as a sensory image, we term the aphasia "*amnesic*." The amnesic does not include the atactic form of aphasia; the former may exist without the latter, in which case the word is readily pronounced when recalled to the acoustic memory.

The study of the disorders of speech associated with insular sclerosis and with the various lesions restricted to the medulla oblongata and pons, brings us accordingly to the probable conclusion *that the infra-cortical regions of the brain contain merely the apparatus requisite for the mechanical execution and connection of the movements subservient to articulate phonation; while the formation of syllables and words, as elements of spoken language, takes place in the cortical substance itself.*

The *cerebellum* is usually credited with the function of immediately regulating the voluntary movements in conformity with sensory impressions. Deiters and Wundt regard the cortex cerebelli as a terminal central expansion, specially designed to serve as a meeting-place for diverticula from the motor and sensory conducting tracts, between which it is, as it were, interposed; so that the motor provinces of the cortex cerebri, on the one hand, and the sensory provinces of the general surface of the body, on the other, are both represented in it.

Luys,[1] even before Jaccoud, had assigned an important part in the production of articulate sounds to the cerebellum. He asserts that disorders of speech have been observed in one-fifth of the total number of cerebellar lesions, these disorders ranging from simple weakness to complete loss of speech, and being of both an asthenic and an atactic kind. He found disorders of speech noted in fifteen out of fifty-six cases of cerebellar disease; in five out of the fifteen there was aphasia. On the post-mortem table, new growths (including tubercles) and hemorrhagic extravasations were discovered.[2] Now, although we cannot dispute the possibility of the cerebellum playing some part or other in the co-ordination of phonic movements and in the rhythm of speech, still the observations we have at our disposal (some of which date from the last century) are ill adapted to serve as a foundation for positive conclusions.

The majority are cases of tumor.[3] Now, cerebellar tumors often exert pressure on the medulla oblongata and pons—organs whose importance for articulation is securely established. Or they may give rise to internal hydrocephalus, often of enormous proportions, which hinders the transmission of motor impulses to the organs of speech, and produces amnesic aphasia by dulling the mental faculties. Neither can we draw satisfactory conclusions from the rare instances in which some disorder of speech was observed to coexist with softening of, or hemorrhage into, the cerebellum, for the records are too imperfect for our purpose.[4] —Of far higher value is Combette's celebrated case of complete deficiency of the cerebellum[5] in an imbecile girl who died in her eleventh year. The cerebellum was only represented by little stumps no bigger than peas; its middle crura (Brückenarme) with the transverse fibres of the pons were entirely wanting. The child, when admitted in her tenth year into an orphanage, was able to answer questions, though her articulation was not very distinct ("nettement"). In the last year of her life, her replies were limited to "oui" and "non." The atrophy was probably a result of some slow destructive change; but Cruveilhier was unable to satisfy himself whether the change had begun and run its course before or after birth. — Dr. Verdelli, of Brescia,[1] has lately published a similar case of atrophy of the cerebellum in an epileptic, aged nineteen. The cerebellum was reduced to the size of a nut; the pons measured two centimetres in its transverse, and a little over one centimetre in its long diameter; the medulla oblongata was barely half as broad as usual. The cerebral hemispheres appeared to be of normal size. The young man, though paralyzed in the legs, was fairly intelligent, joked with the other lads, and followed their conversation. He was late in learning to speak; he stuttered, and was often unable to utter a particular word.—On the other hand, the faculty of speech does not appear to have suffered in the case of rudimentary development of the cerebellum lately described by Otto.[2] Here, it is true, the rudiment was much larger than in the two other cases. It was made up of the normal tissue-elements of the cerebellum.—The retention of the faculty of speech in cases of unilateral atrophy of the cerebellum proves nothing against the importance of this organ in regard to speech; for the functions of the missing half may possibly be fulfilled by a vicarious activity—perhaps associated with hypertrophy—of the other half (Lallement).

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[1] Recherches sur le système nerveux cérébro-spinal. Paris, 1865.

[2] *Benedikt*, Nervenpathologie und Electrotherapie. Leipzig, 1874. Abtheilung I. p. 343.

[3] *Ladame* (Symptomatologie und Diagnostik der Hirngeschwülste. Würzburg, 1865) collected 78 cases of cerebellar tumors, in only 5 of which was any disorder of speech noted.—*A. Ferber* (Beiträge zur Symptomatologie und Diagnose der Kleinhirntumoren. Marburg, 1875) found imperfect mobility of the tongue and disorders of speech frequently mentioned in 32 more recent observations. The two together were observed in 8 of the cases.

[4] Cf. *e. g.*, *Andral*, Clinique médic. III. edition. Vol. V. p. 495.

[5] *Cruveilhier*, Anatomie pathol. Livr. XV. Pl. V.

CHAPTER XIX.

General Account of the Conducting Fibres and Ganglia through which Speech-impulses Pass from the Cortex Cerebri to the Basal Phonic Centre.—The Corpora Quadrigemina and Optic Thalami in their Relation to the Faculty of Speech.—The Channels between the Crusta of the Cerebral Peduncles and the Cerebral Cortex, as made out by Gudden and Veyssière.—Anterior and Posterior Centro-hemispheric Dysarthries of Conduction.—In most Human Brains the Main Current of Centrifugal Impulses in Speaking passes downward through the Left Cerebral Hemisphere, a Side-current through the Right One.—The Connection of the Motor Channels of Speech with the Central Aggregates of Gray Matter.—The Gray Matter of the Corpora Striata in its Relation to Speech.—Broadbent's Hypothesis.—Unequal Value of Existing Clinical Materials for the Localization of the Functions concerned in Speech, according to the Anatomi-

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[1] *Schmidt's* Jahrb. Bd. CLXV. p. 23.

[2] Archiv f. Psychiatrie. Bd. IV. p. 730, und Bd. VI. p. 859.

cal Nature of the Lesions.—Strio-capsular and Strio-nuclear Dysarthries.—The Domain of Dysphasic Troubles begins beyond the Corpora Striata in the Hemispheres proper (Hirnmantel).—The Path traversed by the Articulatory Impulses for Literal Phonation in the Anterior Portion of the Corona Radiata, and its Relation to the Associative and Commissural Channels in the Hemispheres Proper.

Having established the existence of a basal phonic centre, and having endeavored to ascertain the importance of the medulla oblongata, pons and cerebellum in relation to speech, so far as the data we possess enable us to do so, we may proceed to acquaint ourselves with *the ascending and descending, sensory and motor, fibres and ganglia traversed by the impulses concerned in speech on their way between this basal phonic centre and the cortex of the cerebral hemispheres.*

Every sensory and motor province in the body is centrally represented, not merely in the medulla spinalis or oblongata and in the cerebellum, but several times over in the cerebrum itself. Meynert divides the ganglionic masses of the cerebrum into two great regions, one of which receives its fibres through the fillet (Schleife) and tegmentum, the other through the crusta pedunculi cerebralis. The former includes the corpora quadrigemina and optic thalami, which he terms *ganglia of the tegmentum;* the latter, comprising the corpora striata and cortex cerebri, he calls *ganglia of the crusta.* The ganglia of the tegmentum, in all likelihood, subserve only the reflex movements which are determined by sensory impressions, either simple or combined, to form images; the ganglia of the crusta, on the other hand, subserve the voluntary movements which originate in states of consciousness. Ideas, and the volitional motor impulses which traverse the channels of the crusta, are generated in the cell networks of the cortex, the corpora striata assisting in the execution of the movements prompted by the will. The latter are undoubtedly accessory motor organs of volition,[1] though the exact nature of the aid they render to the will is not yet fully understood. Again, the two great groups of ganglia—that of the tegmentum and that of the crusta—are connected together by a special system of fibres; the cortex cerebri is in communication with the tegmental ganglia by means of fibres which convey sensory stimuli from the latter to the former, and autogenous impulses inhibiting reflex action from the former to the latter.

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[1] The basal portion of the corpus striatum seems, moreover, "to stand in a relation to the organs of smell similar to that in which the corpora quadrigemina stand to those of vision, and the optic thalami to those of touch, *i. e.*, to be concerned in the determination of those movements which depend on olfactory impressions."—*Wundt.*

First, then, as regards the *ganglia of the tegmentum*. We may confidently affirm that the *corpora quadrigemina*, though in their optical capacity of the utmost importance for written language, are in nowise concerned with speech.

Concerning the *optic thalami*, we must express ourselves with more reserve. It is true that experiments on animals have not hitherto shown any appreciable failure of motor power or of cutaneous sensibility to follow destruction of the optic thalami[1]; still, some degree of motor disturbance has been observed which seemed to indicate enfeeblement of the muscular sense.[2] This suggests a possible mode in which lesions of the optic thalamus may impair articulation. For the rest, the combined hemiplegia and hemianæsthesia commonly observed to follow hemorrhage into the hinder part of the optic thalamus in the human subject, and with which a variable amount of disturbance of articulation is frequently associated, is not, in all likelihood, an immediate effect of the damage inflicted on the optic thalamus, but results from it indirectly, owing to the pressure of the extravasated blood on the adjoining internal capsule (Meynert and Charcot).

Pelzer[3] has seen a case of apoplexy in which patches of embolic softening were found in the posterior and outer third of both optic thalami, in the lateral part of the corpora quadrigemina, and in both hind lobes of the cerebrum. The only motor trouble observed was *a somewhat hesitating utterance;* the patient was able to put his tongue out straight; he was quite blind, and his memory was impaired. Ought the hesitating utterance in this case to be ascribed to circulatory disturbance in the adjacent motor regions, which must have been implicated in the effects of the plugging of the basilar artery? Or ought we to regard it as a result of enfeeblement of the muscular sense, due to the softening of the thalami?

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[1] *Ferrier* alone asserts (Arch. génér. de méd. October, 1875. p. 503), from experiments on monkeys, that destruction of the optic thalamus causes universal anæsthesia of the opposite half of the body.

[2] Cf. *Nothnagel*, Virchow's Archiv. Bd. LXII.

[3] Berl. klin Wochenschrift, 1872. No. 47.

There are also some older observations on record which warn us against too absolute an expression of opinion concerning the motor insignificance of the optic thalami in man. Huguenin,[1] for example, thinks himself entitled to infer, from an observation published by Duplay,[2] that the facial nerve receives fibres from the optic thalamus. Durand-Fardel, too, records a case[3] which seems to prove that articulate speech may be impaired by a cystiform residue of softening, no bigger than an almond, in the left thalamus. Here, it is true, there does not seem to have been any actual paralysis. "Une gêne assez notable de la parole" was noticed, but no palsy.

Let us now go on to consider *the channels traversed by speech impulses between the crusta and the cortex cerebri.*

Experiments which have recently been carried out on dogs in Vulpian's laboratory, with an ingenious instrument contrived by Veyssière,[4] have taught us a great deal about the exact course of the fibres between the crusta of the cerebral peduncle and the cortex. Moreover, the information gained from the study of circumscribed lesions of the central part of the hemispheres, due to softening and hemorrhage, agrees with the results of experiment.

The great tract to which Burdach gave the name of "internal capsule" contains, in the posterior third of its course, between the optic thalamus and the lenticular nucleus, both motor and sensory fibres. As they pass on towards the periphery of the cerebrum, these fibres separate from each other—the motor ones bending forward, the sensory ones backward. The anterior part of the internal capsule, which lies between the nucleus caudatus and the first segment of the nucleus lenticularis, is purely motor, the sensory fibres being directed towards those convolutions which are situated behind the Sylvian fissure. We may safely conclude with Meynert, that some of these sensory fibres, which ascend from the crusta, bend round directly into the corona radiata of the occipital lobes before reaching the lower edge of the lenticular nucleus, and without entering into any connection with gray matter on their way. These sensory fibres are said to have been traced, in the brain of apes, through the pons and pyramids into the posterior columns of the spinal cord. Whether any of the motor fibres of the crusta which traverse the internal capsule on their way towards the frontal region of the hemisphere, pass likewise into the anterior corona radiata without any intermediate connection with the gray matter of the corpus striatum (either its caudate or its lenticular nucleus), is not equally certain. This, at any rate, is certain: that many, if not all, of the motor fibres of the internal capsule penetrate into the cell-networks of the corpora striata. Further, there can be no question that the motor fasciculi of the crusta are continued downward through the pons and pyramids into the lateral columns of the spinal cord.

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[1] Allg. Pathol. der Krankh. d. Nervensystems. Th. I. Zürich, 1873. p. 186.

[2] Union médic. 1854. Paralysis limited to the cheek, due to circumscribed softening in the optic thalamus.

[3] Traité du ramollissement du cerveau. 1873. p. 371.

[4] *Veyssière*, Sur l'hémianesthésie de cause cérébrale. Thèse. Paris, 1875.—*Lépine*, De la localisation dans les maladies cérébrales. Thèse. Paris, 1875.—*Carville* and *Duret*, Archives de physiologie norm. et patholog. 1875. T. II. p. 352.—*Charcot*, Des localisations dans les maladies cérébrales. Progrès médic. 1875. No. 17, seqq.

These results of anatomical and experimental research are in harmony with those reached by Gudden along an altogether different road. He removes portions of the brain from newly-born animals, allows them to grow up, and then proceeds to study the secondary atrophies resulting from the original injury, with a view to determine the course taken by the different sets of fibres, and the connection of the different centres with one another. His experiments were made on rabbits and dogs. His principal conclusions are embodied in the following brief communication, which he has been good enough to send me at my request.

"In the rabbit the frontal region of the brain extends beyond the limits of the frontal bone. If we remove the superficial layers of the hemisphere where it is protected by the latter bone, we get atrophy of the middle portion of the cerebral peduncle, and very marked wasting of the pyramid (these changes are quite appreciable by the unaided eye). If we slice off the surface of the hemispheres where they are covered by the parietal bone, we get atrophy of the lateral part of the crusta of the cerebral peduncles, and, in a very moderate degree, of the pyramid also. Examination of sections shows, in either case, a partial atrophy of the internal capsule, which is differently localized in the two cases, and is in need of more detailed investigation. I believe I am justified by my inquiries in denying that, in the first case, the corpuscular elements of the corpus striatum undergo atrophy; this, however, is certain: that a multitude of fibres proceeding from the corpus striatum through the internal capsule to the forebrain, disappear without leaving any trace. In the second case, on the other hand, even inspection with the naked eye is enough to prove that the thalamus is considerably reduced in size. This is another point which requires further investigation. I am convinced that if, in some future experiment, I remove a portion of the cortex under the parietal bones, in addition to that which is covered by the frontal bone, I shall get complete atrophy of the pyramid; while, if I remove the convex surface of the middle and hind brain without injuring the anterior strip of the 'subparietal portion of the hemispheres,' I shall find the pyramid not atrophied.

"In the dog the frontal region of the brain is wholly covered by the frontal bone. Removal of its surface-layers is followed by shrinking of the median portion of the crusta of the cerebral peduncle and total atrophy of the pyramid. Examination of sections displays, just as in the rabbit, partial atrophy of the internal capsule. Superficial ablation of the hemisphere underneath the parietal bone is followed by shrinking of the lateral part of the cerebral peduncle; while the two pyramids remain, as nearly as possible, equal in their external diameters."

In connection with the researches of Tuerck and the experiments of Veyssière, Charcot has shown that, in man, blood-clots and patches of softening situated in the hemispheres may give rise either to purely motor paralysis, or to hemiplegia with hemianæsthesia on the opposite side of the body; in the former case the transmission of impulses is interrupted through the anterior, in the latter, through the posterior part of the internal capsule; the interruption being due either to rupture of the fibres, or to their being compressed by changes in their neighborhood (optic thalami, lenticular nuclei, external capsule, etc.). When the interruption is caused by compression, conductivity may be re-established on absorption of the extravasated blood; when due to rupture of fibres, it is permanent. Charcot terms the anterior part of the central region of the hemispheres, damage to which is followed by purely motor hemiplegia, "the domain of the arteriæ lenticulo-striatæ;" the posterior region, damage to which is followed by hemiplegia with hemianæsthesia, he calls the domain of the "arteriæ lenticulo-opticæ;" the parts receiving their blood-supply from these two sets of arterial twigs respectively. Accordingly, when dysarthric imperfections of speech, resulting from interrupted transmission of volitional impulses, are associated with centro-hemispheric hemiplegia, they may be ascribed to lesions of the anterior region when unattended by hemianæsthesia; of the posterior region, when hemianæsthesia exists. Thus, we may have *anterior and posterior centró-hemispheric dysarthries of conduction.* The sooner the defect of speech disappears after the apoplectic stroke, the greater the likelihood of its having been caused by compression; the longer it lasts, the more probably may it be ascribed to rupture of conducting fibres.

Central blood-clots and patches of softening in both hemispheres, which damage the whole of the voluntary motor tract on its way through the right and left internal capsule, necessarily interfere with articulate utterance, and render speech stammering, muttering, or altogether impossible.—R. Bright, for example, saw lasting difficulty of articulation and of deglutition, together with partial paralysis of the limbs, in a case of softening of both corpora striata. In regard to this case, and all other cases of old date, in which one or both of the corpora striata are stated to have been extensively damaged, we shall probably be right in assuming, not the gray nuclei of these bodies only, but the white fibres between them likewise, to have been injured.

But even when the centro-hemispheric or capsular channel for volitional impulses is extensively damaged on one side only, severe stammering or complete loss of articulate utterance may result. Experience has taught us that extensive damage to the left corpus striatum is much more prejudicial to articulation than similar injury to the right one. Blood-clots and patches of softening in the centre of the left hemisphere, followed by right hemiplegia, are much more frequently associated with severe and lasting defects of articulation than corresponding lesions situated in the right hemisphere, and followed by left hemiplegia. Loss of speech, when it occurs in connection with the latter, is usually transient, leaving at worst only a slight impediment of articulation behind.

In a case of softening of the left corpus striatum, Romberg[1] found consciousness unimpaired, while the faculty of speech was totally and irremediably destroyed; there was permanent right hemiplegia; hemianæsthesia and amaurosis were likewise present at first, but disappeared in a few days. Andral,[2] too, records a case in which the left corpus striatum was almost completely softened, the softening not extending beyond it; the intellectual powers were not affected, but the patient's utterance was altogether unintelligible; there was also hemiplegia of the opposite half of the body. The old woman previously referred to, whose case was published by Durand-Fardel, suffered for a whole year from difficulty of speech without paralysis after an apoplectic fit, resulting in the formation of a cyst in the left corpus striatum. She then got softening of the whole of the left corpus striatum, and her speech became altogether unintelligible—"elle bredouillait." Her intelligence did not seem to be impaired. In a right-handed woman of sixty-four, Vulpian found an extensive patch of old softening[3] in the left corpus striatum, occupying the whole of the lenticular nucleus and the middle portion of the nucleus caudatus. She had had an apoplectic stroke five years before her death, followed by permanent right hemiplegia with rigidity. For several months she continued speechless; subsequently, however, she regained the power of expressing her meaning pretty distinctly.

Bateman[4] has published a case in which articulation was severely disturbed by softening of the posterior two-thirds of the right corpus striatum. In our own clinical wards we had an opportunity of seeing a right-handed woman of seventy, who was struck down by apoplexy six weeks before her death. She fell to the ground unconscious. When consciousness returned she was unable to speak, but the power of speaking soon returned. Complete hemiplegia and hemianæsthesia of the left side persisted until death. The tongue was protruded towards the paralyzed side, the mouth was drawn to the right, articulation was rather difficult, but she was able to express herself quite distinctly and intelligibly. A patch of hemorrhagic softening occupied the hinder two-thirds of the right lenticular nucleus; it involved the external capsule between the lenticular nucleus and the claustrum, and the claustrum itself, projecting forward as far as the insular cortex, but without involving either this or the external capsule. The blood must have compressed the internal capsule and the insula when it was first poured out; the permanent effects, however, were due to the destruction of the posterior two-thirds of the lenticular nucleus and the external capsule.

The familiar fact that centro-hemispheric hemiplegia of the right side is often associated with defects of articulation, frequently amounting to total loss of speech, which are at once more lasting and more severe than the slighter and more transient dysarthries occurring with left hemiplegia, proves that *the main current of the centrifugal impulses of speech passes downward through the left cerebral hemisphere.* But since dysarthric troubles—though usually of a trifling kind—are noticed, as a rule, in connection with left hemiplegia likewise, we must conclude that, *besides the main current just referred to, a weaker accessory current is transmitted through the right hemisphere.* We shall see hereafter that this pre-eminence of the left hemisphere is connected with the right-handedness of the majority of mankind, *i. e.*, with the special training of the left cerebral hemisphere for the execution of voluntary movements.

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[1] Loc. cit. p. 946.

[2] Clin. Médicale. Vol. V. p. 327. Cf., too, Obs. XXIII. p. 338. Muttering speech with softening of left corpus striatum.

[3] *Mongie*, De l'aphasie. Paris, 1866. Obs. XVI.

[4] On Aphasia. p. 59.

Persons rendered hemiplegic and speechless by such centro-hemispheric extravasations and softenings are able (as has been pointed out by Trousseau and others), provided there be no cortical complications, to express their thoughts in writing with the sound hand. It is hardly necessary to add that the first attempts of right-handed people to write with the left hand are usually very awkward. Specimens of such writing have been published by Laborde.[1]

We know scarcely anything, unfortunately, about the exact course followed by the motor fibres subservient to articulation, on their long journey from the frontal cortex to the basal phonic centre, or about their connection with the central masses of gray matter.

Even as regards *the medulla oblongata*, on whose intimate structure microscopical research has thrown the most light, our knowledge is exceedingly imperfect. According to Huguenin[2] the will-fibres of the facial and hypoglossal nerves, on their way down from the brain, do not penetrate into the bulbar nuclei, but receive special motor fasciculi from the latter, in conjunction with which they issue from the medulla oblongata to form the trunks of the facial and hypoglossal nerves. He further asserts that the nucleus of the facial receives fibres from that of the abducens likewise, and, as I have already pointed out, from the optic thalamus. Exner's experiments, referred to above, seem to indicate that before a centrifugal impulse from the cerebrum enters the nerve-roots, it has to traverse ganglionic masses in which it is at once delayed and intensified. Again, there is the fact (a detailed consideration of which must for the present be deferred) that the bilaterally symmetrical muscles of the larnyx, tongue, etc., are thrown into contraction simultaneously in speaking, a fact which cannot be explained without assuming the existence of commissures which at some point or other divert the impulse to the opposite side. It is probable, indeed, as I have already observed, that excitor currents flow downward through both hemispheres; the current derived from the left hemisphere being, however, the more powerful and the really operative one, since destruction of the centro-hemispheric channels on the left side is of itself sufficient to annihilate the faculty of speech for a long time, or even permanently. All these considerations taken together compel us to believe that *commissures and collecting ganglia are intercalated somewhere or other below the cerebral peduncles.* They tend, moreover, to enhance the importance of the medulla oblongata, which we have already had occasion on other grounds to recognize, in regard to the function of articulate utterance.

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[1] Bulletin de la société anatomique. VIII. 2me part. p. 386.

[2] Loc. cit. p. 166 and p. 168. *The same*, Ueber die cerebralen Lähmungen des N. facialis. Corresp.-Blatt. f. schweiz. Aertze, 1872. Nos. 7, 8, and 9.

Huguenin views those facial and hypoglossal fibres which are derived from the nuclei as ancillary to reflex movements; contrasting thereby with the volitional fibres derived from the cerebrum. He refers to the fact, which was observed long ago by Bell, Stromeyer, and Romberg,[1] that the power of voluntary movement may be abolished in the area of distribution of the facial and hypoglossal nerves, while reflex movements continue to be executed, and *vice versâ.* He adds an illustrative case of his own.[2]

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[1] *Romberg*, a. a. O. p. 786.

[2] In a boy aged 7 the site of the facial nucleus on the right side was occupied by a tumor as large as a bean. The paralyzed half of the face remained flaccid when he laughed or cried; but it took part in the movements necessary for whistling and clos-

Many authors go so far as to assume the existence of two facial nuclei, one for the orbital, the other for the buccal branches of the nerve

The fibres of the facial decussate, as is well known, in the lower third of the pons. The decussation of the hypoglossal fibres probably takes place in the pons likewise, but we have no precise knowledge on the subject.

Whether the masses of gray matter in the cerebral peduncles take any part in articulate phonation and are connected with the motor tract of speech, must for the present be left an open question. It is clear, however, that any interruption of the motor channels in the peduncles must interfere with articulate utterance.

In the cerebrum the buccal fibres of the facial take a different course from those distributed to the orbit. It is clear that in the hemisphere the facial nerve must break up into a bush-like expansion of diverging fibres, since lesions in various situations are followed by partial palsy of the facial muscles. Concerning the course of the hypoglossal fibres in the hemispheres, we are altogether ignorant.

We may now go on to inquire whether, and in what way, *the gray matter of the corpora striata* takes part in the function of speech.

The ganglia comprehended under the collective name of "corpus striatum," viz., the *nucleus caudatus* and the three segments of the *nucleus lenticularis*, are commonly viewed as organs for those combined movements which are determined by impulses transmitted from the cortex cerebri, *e. g.*, running, leaping, etc. This view is in harmony with the results of the experiments originally made by Magendie, and recently repeated and extended by Nothnagel,[1] which have led to the latter's discovery of a circumscribed nodus cursorius in the caudate nucleus. We are thus brought to regard the corpus striatum as a rendezvous for the fibres coming from the cortex and those going to the pons;

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ure of the eyelids. Speech was "awkward."—Cf. also *Joffroy*, Gazette méd. de Paris. Nos. 41, 42, 44, 46.

[1] Virchow's Archiv. Bd. LVII. p. 27.

the two sets of fibres being connected through cell-networks and nodal points capable of transforming the impulses received from the cortex into combined movements, whose individual elements or constituent acts are in their turn co-ordinately executed by the cell-networks of the medulla oblongata and spinal cord. Of course our knowledge on this point is not absolutely certain and exact.

Moreover, Nothnagel[1] considers himself justified in concluding from his experiments on rabbits, that all the will-fibres, without exception, traverse the lenticular nucleus. This is denied by the French investigators, who attribute the complete annihilation of voluntary movement, which Nothnagel achieved by the injection of caustic solutions into both lenticular nuclei, to a simultaneous destruction of the internal capsule, which, owing to the small size of the lenticular nuclei in the rabbit, might easily have been overlooked on dissection. Nothnagel, however, has lately published a brief statement in which he adheres to his original assertion.[2]

Lastly, Burdon Sanderson[3] affirms that, by electrical stimulation of definite points on the surface of the exposed corpora striata, he has succeeded in producing the very same combined movements as those observed by Hitzig to result from stimulation of definite points in the frontal cortex. But neither Ferrier nor Carville and Duret were able to produce such movements by electrical stimulation of circumscribed areas of the corpus striatum ; they only observed pleurosthotonos on the opposite side of the body. Moreover, Hitzig asserts that the motor points which Sanderson believes himself to have detected in the corpora striata do not correspond in position to those discovered by Hitzig himself in the frontal cortex.

The recorded cases of damage to the corpus striatum in the human subject do not, unfortunately, furnish adequate data for enabling us to decide on the functional significance of its gray nuclei, either singly or collectively. We are seldom informed of the precise extent of the lesion, and whether it was limited to the gray matter, or involved the white fibres as well. According to Charcot, circumscribed lesions of the gray nuclei alone, and lesions confined to the white capsular fasciculi, are both of them followed by hemiplegia on the opposite side of the body. But the paralysis resulting from injury to the gray matter is not so lasting, and therefore less severe, than that which follows injury to the internal capsule, the relatively transient character of the former being due, in all likelihood, to the capacity of the ganglion-cells of the caudate and lenticular nuclei for vicariously fulfilling one another's functions.

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[1] Virchow's Archiv. Bd. LX.

[2] *Ibid.* Bd. LXVII.

[3] Med. Centralblatt. XII. No. 33. 1874.

Broadbent assumes that *the corpora striata are organs in which single words, or even groups of words, are formed and combined as motor acts*, just like the combined movements required for running, leaping, etc. A critical examination of the clinical data we possess does not, however, substantiate this hypothesis. On the contrary, it tends to prove that the co-ordination of letters to form words takes place higher up—in the cortical substance. Broadbent himself admits one difficulty in the way of his hypothesis, viz., that, while disorganization of one corpus striatum—more particularly the left one—interferes with speech only for a time at the outside, destruction of the third left frontal gyrus (whose importance in relation to speech, as a motor act, is supreme) is followed by a more lasting impairment of this function. The transient loss of speech—so far as it consists, not in a mere inability to utter articulate sounds, but in a want of power to form words—which results from grave lesions of the corpus striatum, appears to be readily susceptible of explanation by taking the indirect effects of such lesions into account. They must interfere with the functional activity of those adjacent regions of the cortex which preside over the formation of words, either by squeezing the blood out of them or by setting up collateral fluxion in them. This would afford a simple explanation of the facts, without any of those elaborate attempts at elucidation to which Broadbent has recourse, and which we need not enter upon here.

In the whole of our medical literature, I know of only one case which might be quoted in support of Broadbent's theory; it is only just to add that the case in question was observed by a very eminent physician. It might be quoted to prove that an isolated lesion of the gray matter of the corpus striatum is able to annihilate the power of forming words, without interfering with any other department of volition.

Andral[1] relates that a woman, eighty years of age, lost the faculty of speech quite suddenly (so, at least, her friends said), three years before her admission into La Pitié. The loss of speech was not attended by any other cerebral disturbance. On admission, she was found to be *quite speechless*, unable to utter a single word; but she understood what was said to her, and expressed herself by means of signs. She had the use of all her senses, and perfect control over the movements of her tongue and limbs. Her death was caused by an affection of the heart and bed-sores. Nothing was found in the brain except two old patches of softening, containing a grayish, gruelly matter, and as big as large peas. One of them was situated in the hinder end (the apex) of the left corpus striatum, the other in the centrum ovale of the right hemisphere.

Since lesions of the left cerebral hemisphere are far more dangerous to speech than lesions of the right one, we may fairly attribute the loss of speech in the above case to the small patch of softening in the left corpus striatum. But, as Andral justly remarks, we cannot tell how long the lesion had been there. Both patches may have been more recent than the lesion which caused loss of speech three years before, and which may possibly have eluded observation on the post-mortem table, for we do not always succeed in discovering the anatomical cause of speechlessness after death. The coexistence of the little patch of softening in the left corpus striatum with the symptom in question may not improbably have been an accidental one, for the case is unsupported by any other observations of a similar kind by which its significance might be enhanced.

But although we are compelled to reject the opinion that the corpora striata take part in the formation of words, they may nevertheless participate in the mechanism of literal phonation and the articulation of syllables—so far as the latter consists in the mechanical transition from one muscular attitude to another, and, more particularly, in the "vocalization" of consonants (combination of consonants with vowel-sounds). There is reason to believe that the basal mechanism of articulate phonation is inadequate *per se* for the formation of the literal sounds, and requires the co-operation of the corpora striata. The higher we ascend in the animal series, the less competent for achieving the more elaborate kinds of movement do the infra-hemispheric ganglia of the brain become, the less perfect does the independence of the lower centres appear. Dogs do not seem to be able to run, after their cerebral hemispheres have been removed, though rats are still able to do so. Nay, it remains to be shown that a dog can bark, as a frog can croak, without its cerebrum. Soltmann's[1] experiments, at any rate, fail to prove that puppies, from whose frontal lobes the cortical layer is removed shortly after birth, acquire the power of barking properly as they grow up. One puppy, indeed, of those that survived the operation, learned to run awkwardly in eight weeks' time; and Soltmann mentions, in one place, that it learned to bark, a phenomenon not previously noticed ; in another place, however, he says that in reality it only squeaked like a puppy at birth, but never barked in a manner suitable to its age. There can be no question that the mechanism of articulate phonation is only developed in the infra-cortical motor ganglia under the influence of the cortex ; in fact, as regards the mechanism of speech, the infra-hemispheric centres may be supposed to stand towards the corpora striata in much the same relation as that in which the spinal centres stand towards the corpora striata as regards the mechanism of running in man and the higher mammals. For the correct formation of literal sounds and their mechanical coaptation into syllables, it is essential that the motor or articulatory processes set agoing in the corpora striata by impulses derived from the cortex cerebri, should combine with those that go on in the basal ganglia ; just as the corpora striata and spinal cord have to co-operate in the act of running.

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[1] Clinique médic. Ed. III. Tome V. p. 324. Obs. XVII.

The clinical data now at our disposal do not suffice, unfortunately, to furnish more precise information concerning this relation of the corpora striata to the basal phonic centres. So much is certain, that *lesions of the corpora striata may render articulate utterance stammering to unintelligibility, or may even*

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[1] Jahrb. f. Kinderheilkunde. New Series. IX. p. 106. ff. ; also pp. 119 and 129.

annihilate it altogether; further, that these disorders show themselves more certainly and more severely in proportion as extensive areas of the left corpus striatum are damaged. But it must be left for future inquirers to solve the arduous problem concerning the difference of effect produced by lesions of the white and those of the gray matter, and to discriminate between the functions of the individual components of those two kinds of matter. The problem is a difficult one, because we know from experience that small portions of the gray matter may be destroyed without any apparent disturbance of function, or by disturbance so insignificant in degree as to be readily overlooked. Should the destruction be effected slowly, the capacity of the brain for accommodating itself to gradual changes will also come into play, the work of the injured portions of cerebral matter being transferred to those which are still intact.

This point is admirably illustrated by a very accurate observation of Fuerstner's.[1] In a maid-servant, aged thirty, he found a large part of both lenticular nuclei—more especially the globus pallidus (two inner segments of the nucleus) on both sides—destroyed by symmetrical telangiectatic gliomata. Notwithstanding these lesions, it was only towards the close of the patient's life, after and possibly because of the administration of chloral hydrate, that paretic enfeeblement of the limbs, and to some extent of the trunk also, was observed. Up to that time she had shown signs of maniacal excitement; she had been screaming and singing, running about restlessly, tearing up her clothes, and attacking those about her. On the very day before she died, she answered questions correctly in a low voice.

This one case is enough to show that recorded observations must be employed with the utmost caution in determining the functions of the corpora striata and their constituent parts—the anatomical nature, no less than the position of the lesions, requiring to be taken into account. This is true generally of all attempts to localize the functions subservient to speech in different parts of the brain. The most certain evidence by far is that obtained from cases of hemorrhage and circumscribed softening; that furnished by cases of tumor, sclerosis, and inflammatory deposit is of less value for our purpose. But even in cases of the former class we must draw our inferences very guardedly. We must never forget that, among the earlier effects of bleeding into the substance of the brain, we may have, in addition to the symptoms caused by the localized destruction of tissue, others resulting from pressure on adjacent parts, which may last for days and weeks. In such cases, therefore, the later and more permanent symptoms are the most instructive. In cases of softening, on the other hand, the earlier phenomena resulting from interference with the collateral circulation by thrombosis, embolism, etc., will usually disappear more quickly, and the symptoms that persist after a few hours or days have elapsed may safely be attributed to the localized destruction of tissue.

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[1] Archiv f. Psychiatrie. Bd. VI. p. 344.

I have no doubt that in time we shall succeed, by collecting precise observations of hemorrhage and softening in the corpora striata, in making out the exact function of its constituent parts, and in ascertaining whether any and what differences there may be between the purely strio-capsular and the strio-nuclear dysarthries—points about which we are at present profoundly in the dark.

In the preceding chapter I have pointed out how important are the conclusions we are able to draw from cases of insular sclerosis of the brain as to the localization of the functions concerned in speech. I called attention to the fact that the morbid changes in this disease exhibit a decided preference for the central medullary masses, and more particularly the motor regions of the brain, usually sparing its cortex; further, that they occur in both hemispheres at once. Under such circumstances it is certainly a significant fact that in this disease the mechanism of articulate phonation is alone impaired, and not the actual coaptation of words. A case published by Romberg,[1] in which circumscribed atrophy of the left corpus striatum, probably due to antecedent sclerosis, gave rise to severe dysarthry, is of importance in this connection; for it lends support to the view we have already adopted on other grounds, viz., that the left corpus striatum has more to do with articulation than the right one.

A schoolmaster, aged sixty, had suffered for eighteen months from palsy of the legs without impairment of sensibility. Articulation was hindered from the first. Some time would elapse before he could bring out a word; when he did, his utterance was precipitate and attended by a hissing sound. At a later period his speech became an indistinct and all but unintelligible muttering, though his mental faculties remained unclouded and his tongue freely movable. Schlemm found the left corpus striatum wasted, the medulla oblongata normal. The vertebral canal was not opened, so that the cause of the paraplegia was not made out; there may possibly have been sclerosis of the lateral columns of the cord. In this case, just as in the insular variety of sclerosis, there was stammering and muttering utterance without any apparent coarse impediment to the locomotion of the tongue.

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[1] Loc. cit. p. 945.

Tumors of the corpora striata very often interfere with articulation; but there is not much help to be got from these cases in localizing the functions concerned in speech. According to Ladame, of all cerebral tumors, those situated in the pons and corpora striata are most often attended by disorders of speech: the patient's utterance has frequently been noticed to be slow and stammering; impediments of speech depending on impairment of the intelligence and of the memory for words have likewise been observed. The latter fact is readily explained by the pressure of large and rapidly-growing tumors on surrounding parts. Conversely, when the growth of a tumor is gradual, so that it rather displaces than destroys neighboring parts, allowing them time to yield or take on one another's functions, the symptoms invariably caused by destructive processes in the same region may altogether fail to appear.

Confining ourselves to such cases as are suited for critical valuation, both on account of the anatomical nature of the lesion, and because they have been recorded with sufficient exactness, we arrive at the following as our principal conclusion: that *the corpora striata form the uppermost limit of that region within which destruction of cerebral tissue gives rise to nothing more than simple dysarthry in the form of retarded or precipitate and "scanning" utterance, or of stammering and muttering, or even of that total loss of speech which is due solely to annihilation of the mechanism subservient to literal phonation.* As soon as we enter the domain of the cortex proper, including the radiating medullary matter which is traversed by the great associative and commissural fasciculi of the cortical masses, we find ourselves in a region, injury to which is followed by a variety of added disorders of speech involving the actual function of constructing words. This great region, to the consideration of which we are now about to address ourselves, we shall term the "domain of the hemispheres proper" (Hirnmantel), distinguishing between their white and their gray regions.

The functions of the cerebral cortex in relation to speech, and the disorders of speech that result from cortical lesions, will be considered in subsequent chapters. There are only two points into which we need enter at present. We have already learned that the motor fibres of the crusta pass upward through the internal capsule and corpus striatum towards the frontal region of the hemisphere, where they connect themselves with the anterior portion of the corona radiata; the great majority reaching this indirectly through the intervening gray matter of the corpus striatum, while some may possibly enter it directly. The sensory fibres of the crusta certainly pass, for the most part, directly into the hinder part of the corona radiata; whether some fraction of them may not previously enter into connection with the gray matter of the claustrum, and possibly with the hinder part of the lenticular nucleus also, must for the present remain an open question. We must now enquire whether clinical observation furnishes any data capable of throwing light on the course of the articulatory channel for literal phonation in the anterior part of the corona radiata, or of the sensory tract ancillary to speech in its posterior part.

A case[1] that occurred in my own wards proves conclusively that small and sharply circumscribed patches of softening in the anterior part of the corona radiata may cause hemiplegic paresis with disturbance of literal articulation.

In a right-handed man, aged forty-two, a single patch of softening, no bigger than a bean, in the white medullary substance of the right frontal lobe between the corpus striatum and the cortex, gave rise to paresis of the left angle of the mouth and left arm, together with slight awkwardness in the production of articulate sounds. We did not succeed in making out to our satisfaction whether the mobility of the tongue was in any way impaired.

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[1] *Frei*, Archiv f. Psych. Bd. VI. p. 327.

In the above case the accessory current of articulatory impulses through the right hemisphere had evidently been interfered with to some extent, but the power of forming words was not impaired. It would be interesting to ascertain whether similar limited destructions of white medullary matter between the the corpus striatum and frontal cortex of the left hemisphere, through which the main current of articulatory impulses flows, would also give rise to a purely literal disorder of articulation, or whether the formation of words would be interfered with, and when. This would throw light on the exact distribution of the motor projection channels of speech among the co-ordinating and associative channels of the arching fibres which connect the convolutions with one another and assist in the formation of words. A case recorded by Farge proves *that lesions of the white substance of the cerebral hemisphere alone, in the neighborhood of the third left frontal convolution, may, without any simultaneous injury to the gray matter of the cortex, disturb the power of forming words.*

The patient was paralyzed on the right side. The only words he uttered of his own accord were "Ah, si, ah!" "Ah, bon sens de Dieu!" The latter phrase he employed to express pain. To questions requiring a simply negative or affirmative answer, his replies were correct enough: "Ah, oui!" or "Ah, non!" When made to repeat words uttered in his presence, he succeeded in the attempt when the words did not exceed two in number; when there were more than two he would only repeat the two first. The words he pronounced were spoken in a normal tone of voice and well articulated. After his death a careful examination of his brain, both with the naked eye and with the microscope, showed that the third left frontal convolution was healthy, while the adjacent white substance contained a spot of gruelly softening as large as a small egg.

In this case, accordingly, there was true aphasia. Popham[1] has placed a similar one on record. In a case of Jaccoud's,[2] too, there seems to have been aphasia in connection with a lesion situated in this region.

After an apoplectic fit, the patient was found to have right facial paralysis without palsy of the limbs, but with loss of speech. This passed away in a few days. After death, two small hemorrhagic cysts were found in the white substance near the third left frontal gyrus. In this case the effects may perhaps have been due merely to compression of the gyrus.

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[1] Gazette hebdom. 1865. No. 44. [2] *Bateman*, On Aphasia, p. 37.

[3] *Dieulafoy*, Gaz. des hôpitaux. 1867. No. 58.

We have no certain knowledge of the place where those motor fibres of the corona radiata which are subservient to speech enter the cortical convolutions; we know still less about the way in which they are connected with the cell-networks of the cortex.

According to Broadbent, the fibres derived from the cerebral peduncle and the central ganglia proceed, for the most part, to the margin of the Sylvian fissure; while the fibres of the corpus callosum betake themselves, for the most part, to the margin of the longitudinal fissure. He is of opinion that many gyri—*e. g.*, those of the insula—receive neither peduncular nor callosal fibres, and are, therefore, connected in an indirect way only, through other convolutions, with the central ganglia, the peduncle, and the corpus callosum. He believes this class of gyri to stand latest in the order of evolution, to be undeveloped in the brain of monkeys, and probably to subserve psychical functions alone.

On the other hand, it is certain that apart from the motor and sensory fibres penetrating into the cortex from the cerebral peduncles and ganglia of the caudex cerebri—apart from the callosa fibres connecting the hemispheres together—there exist a multitude of arcuate fibres which traverse the *white substance* of the cerebral hemispheres for the most part in a longitudinal direction, and serve to unite the various convolutions of the cortex with one another; the more superficial fibres connecting neighboring points, the deeper ones, such as are farther apart; these are the "associative fasciculi" of Meynert, to which reference has been already made.

The great task of the future—to unravel the tangled paths of feeling, thought, will, and action—makes us feel giddy with our present inadequate power of insight. Yet, even were this task accomplished, we should only have made one step towards a knowledge of the labyrinthine arrangement of fibres in the brain. For the cortex itself is traversed in all directions by countless millions of fibres, uniting the corpuscular elements into innumerable networks, and connecting them with the cell-networks of the ganglia situated lower down in the brain and cord, and even with those ganglionic centres which lie outside the cerebro-spinal axis in the various organs of the body.

CHAPTER XX.

Sensory Channels of Language.—Meynert's "Tone-Field."—Central Laboratory of Word-Images.—The Problem of the Elaboration of Elementary Sensations into Perceptions with Reference to the Localization of the Mind.—Relation of Consciousness to the Psychical Functions in General and to Sensation in Particular.—Latent Consciousness, Personal Consciousness (Consciousness of the Ego), and Self-Consciousness.—Lower or Instinctive, and Higher or Intelligent Judgment.—Relation of the Mechanical to the Mental (Psychical) Work of the Nervous System.—Excito-motor Faculty, Goltz's Faculty of Adaptation, Psycho motor Faculty, or, Sensory-motor Reflex Action, Percepto-motor Reflex Action, and Ideo-motor Reflex Action or Free-Will.—Discursive and Intuitive Thought.—Explanation of the Marvels of Language by the Laws of Organic Evolution, and the Mechanical Principle of Reflex Movement Inherent in all parts of the Nervous System, including the Cortex Cerebri.

The *channel for the sensory impressions of spoken words has its beginning in the peripheral expansion of the acoustic nerve;* that *for written words in the retina.* The former is continued into the acoustic nuclei of the medulla oblongata; the latter passes along the optic nerves and tracts to the gray matter of the anterior corpora quadrigemina which are believed to be the actual optic nuclei. True, some fibres pass from the optic tracts to the corpora geniculata and the pulvinar thalami; but the relation of these fibres to the act of vision is not clearly made out; they do not waste after destruction of the eye-balls in newly-born animals (Gudden). There can be no doubt that the nuclei of the optic tracts in the corpora quadrigemina and optic thalami are further connected, on the one hand, with the occipital cortex through the fibres of the corona radiata; on the other, with the spinal cord through the tegmentum and fillet (Schleife). But the course of the individual fibres has not yet been accurately traced;[1] and we are still ignorant of the locality where the semidecussation of the fibres in the optic tract is completed in the brain.[1] As regards the fibres of the acoustic nerve, all attempts hitherto made to trace direct acoustic channels between the medulla oblongata and the cerebrum, have failed. At the present moment, Meynert is actually of opinion that the major part of those fibres reach the cerebrum circuitously through the cerebellum. At one time[2] he thought he had succeeded in following a bundle of acoustic fibres from the inner acoustic nucleus through the tegmentum as far as the claustrum and into the cortex of the island of Reil. On this supposed discovery he grounded his hypothesis that the Sylvian fissure was "the tone-field of language;" a hypothesis which was very favorably received. Unfortunately, however, it has lost its chief claim to consideration since its conscientious author found himself obliged to withdraw his original statement concerning the acoustic origin of the fasciculus in question.

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[1] *Hitzig* (Centralblatt f. d. med. Wiss. 1874. p. 548), by removing slices of brain-matter in the region of the hind-lobe, caused blindness of the opposite eye with dilatation of the pupil.—Cf., however, the experiments of *Goltz* (to be referred to in Chap.

It is of the utmost general interest to ascertain at what point in the sensory tract the optic and acoustic images, resulting from co-ordination of the impressions on the peripheral expansions of the optic and acoustic nerves, are perceived. For our special purpose, the question may be thrown into a narrower shape: Where are the images of spoken and written words, employed as symbols to represent states of consciousness, created? This question must be kept rigorously distinct from another one—Where are those images interpreted and understood as symbols? The comprehension of words, *i. e.*, the connection of verbal images with their corresponding thoughts, undoubtedly takes place in the cortex cerebri, and there only; but it does not necessarily follow that the images themselves originate in the cortex. That they may do so is possible, but not certain. Just as a word, in its motor aspect, consists of a series of individual movements co-ordinated together in the

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XXI.), according to which mutilation of the cortex, either of the anterior or posterior lobes, gives rise to blindness, provided only it be sufficiently extensive.

[1] Cf. *Charcot's* ingenious essay in the Progrès méd. 1875. Nos. 35 and 49. Likewise *Jastrowitz*, Arch. f. Psych. VI. p. 616.

[2] Wien. Med. Jahrb. Bd. XII. 1866. p. 152.

order of their increasing complexity, so it may be that in its sensory aspect a word is made up of a series of elementary sensations, co-ordinated from below upward into more and more highly complex forms. The power to perceive words as sensory images or combinations may be lost without any impairment of hearing or of sight, and without any appreciable, or, at any rate, considerable failure of intellect. Such disorders of speech, which we shall have to describe more fully hereafter as *word-blindness* and *word-deafness*, prove unquestionably: (1) that the localities where sounds, or lines and dots, are perceived, are not identical with those in which sounds are co-ordinated into and recognized as phonic images, lines and dots as graphic images; (2) that the laboratory for phonic and written images is not the same as that for conceptual states of consciousness. Unfortunately, the experimental and clinico-anatomical data which might illuminate this obscure subject are still wanting. We are thrown back on generalities. One thing, however, is clear enough from what has been already said: even if we suppose word-images to be generated in the cortex, we cannot believe them to be generated in the same regions of the cortex as those in which conceptions originate. This, of course, is quite compatible with the view that they may both originate in the same convolutions, but in different cell-networks.[1]

If we pass on from these preliminary considerations to the problem of the elaboration of the peripheric sensory impressions into word-images, we must set out with a clear idea of *the relation of consciousness to the psychical functions in general, and to sensation in particular*. This is the key-stone of the enquiry concerning the localization of the psychical activities generally, and concerning the place where elementary sensations are summed up into images, in particular.

It is self-evident that psychical (mental) activity must exist wherever sensations are perceived and judgments arrived at; but it is difficult to determine the exact point at which sensation and judgment begin. The cause of the difficulty is this: the only immediate proof we have of the fact that we perceive and judge is derived from our consciousness of perceiving and judging; but that others perceive and judge we can only infer, with more or less of probability, from the similarity between their actions and our own. This leaves a wide field open for arbitrary interpretation, and one man will see psychical activity where another sees only mechanical work.

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[1] Goltz's paper on the effects of mutilation of the cerebrum did not reach me till I had completed this chapter (Archiv für Physiologie. Bd. XIII.). His experiments on dogs throw some light on the points under consideration. I shall therefore return to the subject in the ensuing chapter (XXI.).

From the physiological point of view, none of the mechanical work done by the nervous system can be regarded as a manifestation of psychic (mental) force. On the contrary, *mechanical and psychical activity are joint effects of the excitation of nervous matter, which draws its supplies of energy from the common storehouse of the universe.* Inasmuch as a mental state invariably precedes any movement of the limbs in consciousness, as the particular movement invariably corresponds to the antecedent mental state, our movements appear to ourselves to be carried out by the mind (Seele). In reality, however, both sensation and thought originate in the very same molecular changes in our nervous matter as those in which our mechanical activity has its rise. To reconcile this seeming contradiction between the consciousness of free will, on the one hand, and the dependence of all the mechanical operations of the mind on the material processes of nerve-stimulation, on the other, is the business, not of physiology, but of metaphysics.

The discovery of *the reflex mechanism of the spinal cord* proved that a large proportion, at any rate, of the acts which had previously been attributed to free volition were really due to a mechanical principle inherent in the nervous matter. The spinal cord, after its separation from the brain, is still able to execute a variety of orderly movements; but, whereas these movements were previously appreciated by the cerebrum as direct emanations from the conscious Ego, they now no longer receive any immediate recognition in consciousness. Hence, spinal reflex acts ceased to be viewed as psychical phenomena at all, and were ascribed to an "excito-motor" mechanism in the cord itself; the seat of psychical activity being thus shifted into some region of the brain, above the spinal cord.

But the term "*excito-motor*," as applied to reflex acts, never achieved a universal popularity; the term "*sensori-motor*" was preferred to it. The latter is based on the old doctrine that sensation—a psychical act—is the mainspring of reflex movements—a doctrine which, as physiologists, we are bound to repudiate. But, while we deny that spinal movements are generated by sensations, we do not deny that they may be attended by sensations; nay, we are of opinion that this always happens. The fact that we cease to be directly conscious of spinal reflex acts, when the cord has been separated from the higher centres, is no proof that the cord is destitute of sensibility and of the consciousness which is indissolubly bound up with sensibility. It only proves that the consciousness of the Ego (personal consciousness), in which all psychical phenomena are synthetically organized, resides, not in the cord, but in the brain.

What is commonly understood by the word "consciousness" is merely one of the forms in which consciousness manifests itself. We understand by it that illuminated area in the field of vision of the Ego—not capable of being more precisely defined—across which sensations and mental judgments follow one another successively in linear order. We are only conscious of a sensation or a mental judgment when they happen to cross the threshold of this "*personal consciousness*." Daily experience shows us, however, that sensations and judgments are continually taking place below this threshold. By attending to our mental processes we are able at any moment to raise a multitude of them, which would otherwise have remained hidden, into the field of consciousness. For this reason, and also because we are unable to conceive of sensations and judgments taking place unconsciously, we are driven to assume the existence of a latent consciousness, which is revealed only in the field of vision of the Ego. What we term "unconscious" sensation and "unconscious" judgment are only relatively unconscious processes, differing from those we call "conscious" only in taking place unnoticed by the Ego, either because the Ego does not attend to them, or because they are altogether beyond the limits of his field of vision. *Personal consciousness* (consciousness of the Ego), which belongs of right to every organism endowed with mental faculties and individuality, must be distinguished, further, from that supreme degree of consciousness peculiar to man which we may term *self-consciousness.* We are aware of our own consciousness, and raise it to the level of an abstract conception; this process of abstract reasoning taking place only in the cerebral cortex of man, whose powers of thought are far advanced.

Accordingly, there is nothing to prevent us from assuming that the spinal reflex acts accomplished by the cord, after its separation from the brain, are attended by sensation and consciousness. Admitting their connection with sensation, we need not scruple to term them "sensori-motor" acts, and we must agree with Pflueger in ascribing a "soul" (Seele) even to the spinal cord. He grounds his opinion on the purposeful character of many of the spinal reflex acts in the lower vertebrata; these seem to him to be explicable only on the assumption of a choice of movements determined by mental judgments. For it is not the purposeful character of the spinal acts in itself, but the proof that they are the manifestation of an "adaptive principle" in the cord, which must be our warrant for their psychical nature. Goltz, it is true, was brought by his experiments on the spinal reflex actions of the frog to conclusions different from those arrived at by Pflueger. He does not think the evidence for the existence of a "spinal soul" sufficient. He showed that even the most apparently rational of the spinal reflex acts might be accounted for by a suitably organized mechanism in the cord, without any need for assuming them to be an outcome of mental judgments. But in proving this, Goltz has by no means proved the further proposition, that no mental judgments at all are arrived at in the cord. To prove that spinal reflex acts are not attended by mental judgments, it would be necessary to prove that voluntary acts originating in deliberate choice are immediate mechanical effects of psychic force and not mediately executed through the organic mechanism of the brain. But this does not admit of proof; on the contrary, we are driven to conclude that all movements issuing from the brain ought to be referred to the operation of a general principle of reflex mechanism.

The experiments of physiologists have shown that the higher we ascend in the ganglionic system of the cerebro-spinal axis of vertebrates, the more complex are the movements discharged by internal and external stimuli. If we remove the cerebrum up to, or even including, the optic thalami, we see the play of movements continue to be so skilfully adapted to the environment that such adaptation seems possible only on the hypothesis that it is guided by mental judgment. Animals thus mutilated are still able to run, to jump, to swim, to fly. And these actions are performed not merely by such animals as bring the needful faculties into the world with them, but by such as have to acquire them after birth. They adapt those complex movements to surrounding conditions with a skilled choice which can only be ascribed to mind. The brainless fish will avoid obstacles while swimming by turning now to one side, now to another (Vulpian). The brainless frog remains quiet; but if we pinch him, he will jump, avoiding any object in his way; if one of his legs has previously been attached to his belly, he will creep round the obstacle (Goltz). Brainless birds and mammals, indeed, are unable to avoid obstacles when flying, running, or jumping, like fishes and frogs; they knock against them. The lower centres in these more highly-organized vertebrates are not so independent as this; but they are still in some degree independent. The brainless pigeon, when thrown into the air, will fly; it will wink if a finger be brought close to its eye (Vulpian); it will follow the movements of a lighted candle with its head (Longet); it will often draw its head back if a light be suddenly brought close to it (Vulpian). Brainless rats will leap away when they hear a cat. Young rabbits deprived of a brain will run a few steps when pinched; sometimes they will do it of their own accord, prompted, in all likelihood, by some inward pain (Vulpian).

The frog which (when one leg has been stitched to its belly) crawls round an obstacle in attempting to escape, acts just like a man who perceives and reasons, and not like a mere machine. The bird which closes its eye on the approach of a finger, performs a reflex act of which man only becomes capable after he has learned to see objects, and to judge of their movements.[1]

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[1] According to *Sigismund*, this power is not acquired till about the fourteenth or sixteenth week after birth.

The behavior of the rat which springs away on hearing the noise made by a cat, is like that of a man who has learned by a mental process to establish a reflex connection between certain acoustic images and certain purposeful movements. Since we cannot employ the language of animals to denote what goes on in them, we are obliged to employ the human terms by which we denote similar phenomena in ourselves.

We speak of "*movements adapted in purposeful fashion to surrounding circumstances, by means of perceptions which are associated with mental judgments.*" It is true, as Goltz points out, that these "percepto-motor" reflex acts still exhibit a high degree of automatism. The movements are executed with machine-like precision, without hesitation or delay. But this proves nothing against the participation of the mind in their execution, since the latter is always alike mechanical. It proves merely that a precise movement is associated with a precise perception and a precise mental judgment. It is in this respect that such movements differ from voluntary movements; the latter originate, in animals, from the conflict of elementary states of consciousness—in man, from that of higher abstractions; a conflict, to which we give the name of deliberation, and which issues in decision and action.—As every child must have a name, we may call that form of mental judgment which manifests itself in such reflex movements as originate in the infra-cortical ganglia, and are adapted to simple perceptions, a "lower" or "sensual judgment," contrasting it with that "higher judgment of the cortical intelligence" which is associated with a conflict of ideas. The former may also be termed "instinctive," since it is prone to pass from generation to generation by inheritance, while the growth of the latter is connected with the experience of the individual.

We need not scruple, even in the human subject, to localize the mental faculty which elaborates sensations into images and adapts complicated movements to those images by means of an instinctive judgment, either wholly or principally in the infra-cortical ganglionic apparatus. The study of hallucinations, dream-life, catalepsy, chorea magna, and somnambulism, affords indications of a certain autonomy in the infra-cortical sensory and motor ganglia. True, we should not be justified in summarily transferring the results of experiments on frogs and birds to the human subject. For in man, the greatly developed cerebrum enjoys a degree of supremacy over the basal ganglia and the cortex, together with the corpus striatum over the remaining parts of the cerebrum, which has no anologue even in the most intelligent of the mammalia.[1] Still, the type of cerebral architecture is, in all essentials, the same throughout the vertebrate series, and the same principle of stimulation, sensation, and reflex action holds good throughout. If the "*divisibility of the mind*" (Goltz) is established in the case of animals, it must be true of man also. Psychical sensation exists throughout the entire nervous system, including even its peripheral ganglia; the perception of images, and the faculty of adapting complicated movements (running, jumping, etc.) to those images, makes its first appearance in the intra-cranial ganglia; the will, guided by abstractions of a higher order, is associated with the cortex cerebri. Every department of the nervous system fulfils a psychical function of its own; and the higher we ascend towards the cerebral cortex the more faculties are aggregated for the performance of more and more complex mental operations. *The soul, though it ultimately feels and conceives itself to be a self-conscious unity, is really composite.*

Accordingly, it would be a mistake to suppose that mental activity is limited to the brain or the cortex, while all the processes that go on at a lower level are purely mechanical. The nervous system, as a whole, including its uppermost termination in the cortex cerebri, is at once a mechanical apparatus and an organ of mind. Goltz, whose researches on the psychical functions of the nerve-centres are so ingenious, does not venture to give any precise definition of the nature of the psychic force manifested by the basal ganglia of the frog; he terms the faculty by which, in the execution of reflex acts, complex movements are adapted to perceptions, a "central faculty of adaptation" (centrales Anpassungsvermögen). This term, it is true, does not prejudge the question; but it does not help us much, for it expresses nothing definite. For, strictly speaking, all reflex movements, and even voluntary movements, are adapted to sensory phenomena. In the case of a simple reflex act, such, *e. g.*, as closure of the eyelids against light, the movement corresponds to an unformed or elementary sensation, and the diameter of the pupil is very accurately adapted to the intensity of the luminous stimulus or sensation. In the case of those "percepto-motor" reflex acts (Anschauungs-reflexe), which Goltz has before his mind, *e. g.*, closure of the eyelids against a threatened contact with the conjunctiva, the movement corresponds to a reasoned perception (beurtheilte Anschauung), such as originates in the recognition of an image, *e. g.*, of an object approaching the eye. Finally, in the case of voluntary movements, their character is determined by a whole series of images, or, more correctly, of states of consciousness abstracted from those images. Accordingly, we see that the "faculty of adaptation" is of a very general kind, belonging to all nerve-centres alike, and quite as much mental as mechanical. The term is merely denotative—it explains nothing; it connotes nothing as to the psychical or the mechanical nature of the forces and arrangements which determine the phenomena under consideration; it is concerned solely with the form assumed by the effect of the forces in operation.

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[1] *Soltmann's* assertion (loc. cit. p. 105 *seqq.*) that puppies which survived the removal of the cortical layers of one or both frontal lobes succeeded in learning to run (though late and awkwardly), and to eat their food, shows how little, even in so intelligent a creature as the dog, the cortex contributes to the motor activities of the infra-cortical parts of the brain. We cannot unconditionally reason even from the dog to man. The loss of the cerebral cortex and the corpus striatum of one hemisphere produces only hemiparesis in the dog, while in man it gives rise to hemiplegia.

Neither do I see any advantage to be gained from the use of the term "psycho-motor" to denote voluntary movements as contrasted with simple and percepto-motor reflex acts. The mind, according to my view, has just as much and just as little to do with reflex as with voluntary acts. At bottom it is neither sensations nor perceptions, nor yet states of consciousness, that are the mechanical cause of movement; but always the physico-chemical potential energies that are set free in the sensori-motor centres appropriated to sensation, perception, and ideation, by physiological stimuli. We must either admit reflex acts into the psycho-motor group, or we must exclude voluntary movements from it. Under such circumstances, it appears to me to be all one whether we speak of psycho-motor or voluntary movements, of responsive movements in Goltz's sense or percepto-motor reflex acts, or, lastly, of excito-motor or sensori-motor reflex acts. The essential point in all these enquiries is to determine the special psychical phenomena and motor effects, as well as the internal processes of stimulation, that are associated with the objective activity of each individual nervous centre. If we desire to keep on strictly physiological ground, we must speak only of spinal, bulbar, cerebellar, strio-cerebral, cerebro-cortical movements, etc. But the time for this is not yet ripe, since physiology has only just begun to use right methods for the solution of such problems.

A needless difficulty has been imported into the question of the relation of the soul to the mechanical work of the nervous system on the one hand, to the processes of stimulation in the nervous matter on the other, by the assumption that activity is psychical only when it is immediately realized in the personal consciousness. The fact that only the smallest fraction of even the highest intellectual work takes place in the light of the conscious Ego was overlooked. When the child first begins to think, every perception furnished by its fresh sensations comes before its consciousness. But as we get more practised in thinking, the images of sense grow fainter, *i. e.*, they sink below the threshold of personal consciousness; their place is taken by concepts clothed in words, and the act of thought gains thereby in rapidity and precision. Yet more, many of the states of consciousness, which were at one time knowingly traversed by thought, are subsequently omitted altogether, and are no longer raised into the field of consciousness. The association of ideas is abbreviated; thought, in the words of Lazarus, becomes condensed—intuitive takes the place of discursive thinking. If we are well trained in the matters of logic and psychology, we may be able to test the accuracy of our intuitions by retranslating them into the discursive form and raising our unconscious judgments into consciousness. But we cannot always do this completely, even as regards our conceptions; still less as regards the perceptions obtained on a lower level of psychical activity, with which we operate when thinking. For instance, if we take the perception of space, it is possible to show, by means of experiments on persons born blind and restored to sight by operation,[1] that we probably bring with us into the world only the perception of space of two dimensions, the perception of depth having to be acquired by trial-movements, for whose comprehension a considerable degree of physiological and psychological insight is essential; notwithstanding which, the perception of depth is realized by us in our thinking and acting as though it were some immediate appurtenance of consciousness, as though it were an inborn feeling of instinctive certainty. And even the most abstract kinds of thinking by means of words and formulæ, such, *e. g.*, as are used in mathematics, go on in accordance with rules painfully acquired and subsequently forgotten, *i. e.*, shut out of consciousness, and persisting merely as a "sense of language," "mathematical sense," etc. The assertion that only conscious activity is psychical would imply that some of the most highly-gifted brains, whose intuitions appear so marvellous to us ordinary people, are really the most devoid of reasoning power. A Phidias and a Raphael, a Goethe and a Shakspeare, a Newton and a Leibnitz, would be mere automata. Were mind only then mind, when conscious of its own activity, it would—since the Ego is only capable of realizing sensations and cognitions one by one in linear succession—always exist merely as this or that sensation or cognition entering at the moment into consciousness. But this would be opposed to the fact that our Ego is made up of an enormous aggregate of sensations, images, and cognitions, all of which it embraces and fuses into unity. The conscious manifestations of our individuality never originate in this or that single conscious sensation, perception or cognition, but always in a fused totality of all of them, slumbering in memory, lying hid somewhere or other, vibrating during thought, but only recalled into personal consciousness in very small proportion.

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[1] Cf. the latest observation of this kind by *Hirschberg*, Graefe's Archiv. 1875. Heft I. p. 23.

But if we are obliged to admit that even the highest of our mental operations are to a great extent unconscious, we can scarcely doubt that those on lower levels may be so likewise. In fact, we are driven to conclude that sensation and perception only become "conscious," in the vulgar sense of the word, under definite organic conditions peculiar to the cerebrum. Those conditions are: structural perfection of the conducting fibres and of the ganglia which connect them on the one hand —the orderly occurrence of the material processes comprised under the collective name of "stimulation," on the other.

We find this doctrine in harmony with the evolution of consciousness in the history of our globe and of the human race, with the growth of mental life in the animal series and in the individual man. Wherever we turn our eyes, we find unconscious mental activity undergoing progressive elevation into conscious forms. The unfolding of the mental powers in the animal world goes hand in hand with the ganglionic differentiation of the nervous system. For the intelligent cognition of phenomena, the maximum development of the cerebrum and its cortex, such as we find in man, is requisite. But even homo sapiens is at first, in the impregnated ovum, nothing more than a punctum saliens; it is not till the fifth month that common sensation manifests itself by kicks and pushes; when he enters the world to begin an independent existence, his senses are not all in working order; he is quite deaf, and his visual perceptions are limited to vague impressions of light and darkness, while he has already gathered a harvest of distinct tactile and gustatory impressions in his mother's womb.[1]

The development of the central nervous system in man tends likewise to confirm our theory. The histological investigation of the brain and spinal cord at different stages of their growth shows that the central fibre-systems are built up in succession. The fatty degeneration of the glia-cells, first recognized as an incident of normal development by Jastrowitz, as also the formation of medullary sheaths associated with it, are far from being complete at birth; *they are governed by a principle which amounts to a systematic differentiation of the central fibre-masses* (Flechsig[1]). *Those fibres which subordinate the spinal cord to the volitional centres are the last to be developed.* Moreover, they attain a higher grade of development in man than in any other animal. *The fibre-systems concerned in voluntary movement are the key-stone of the fibre-structure in the medulla oblongata and spinal cord* (Flechsig).

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[1] Cf. my researches on the Mental Life of the Newly-born Infant. 1859. Also *Troeltsch*, Lehrbuch d. Ohrenheilk. Leipzig, 1873. pp. 22 and 23.

Soltmann has shown that the results of physiological experiment are in harmony with these anatomical facts. In the newly-born puppy about ten days have to elapse before the electric excitability of the cortex cerebri (or, more correctly, of the medullary fibres that issue from it) is developed, while that of the internal capsule is present from the first.

The mind, accordingly, begins its work with sensory activity, and its earliest (purely reflex) motor acts are definitely prescribed to it by the inborn anatomical and physiological interconnection of the ganglionic cell-networks in the peripheral, bulbo-spinal, and basal centres. Shortly after birth, the waves of stimulation, ever extending in wider circles, rise as high as the brain. Perceptions and judgments are continually added in growing abundance and increasing differentiation. True, movements still follow stimuli with automatic precision; but they are susceptible of purposeful modifications in form under the controlling influence of images composed of sensory impressions, and by virtue of assured judgment. The cortex cerebri begins to share in the excitement of the lower centres; its vibrations, gentle at first, increase progressively in amplitude; the instinctive mind becomes intelligent, fulfilling movements with intention, and controlling the reflex activity of the infra-cortical centres; in other words, it becomes endowed with will. Combinations, at first few and simple, but increasing in number and complexity, become associated in the vast cell-networks of the cortex. Bain has calculated with much ingenuity, that the number of combinations rendered possible by the wealth of the cortex in cells capable of receiving impressions and in connecting fibres, is sufficient for the total number of states of consciousness existing in the most powerful memory and the most highly-gifted intellect. There is room enough in the physiological seat of the intelligence for all the impressions derived from the world of sense; there is room enough for the whole motor key-board of the human will. The problem of constructing a laboratory of intelligence, and of will determined by intelligent motives, out of organic matter, is thus solved in marvellous fashion. True, the marvel does not begin here; it begins at the point where the state of excitement induced by stimulation in a material nerve is converted into an immaterial sensation.

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[1] Die Leitungsbahnen im Gehirn und Rückenmark des Menschen. Leipzig, 1876.

According to the description I have just given, sensation constitutes the elementary psychical function of the nervous system. Its specific energies are determined, first, by the nature of the peripheral sense-organs with which the nervous apparatus is connected; secondly, by the connections formed by the conducting fibres in the central ganglia; just as motor acts are determined, on the one hand, by the muscles and limbs to which the motor fibres are distributed; on the other, by the central connections of those fibres. Perceptions and states of consciousness are not elementary, but compound processes, originating in sensations and movements; and even in personal consciousness (consciousness of the Ego) we can see nothing more than a higher synthetic unity of psychical phenomena.

By adopting these fundamental principles, we are enabled to understand many of the marvels of language and to subordinate them to the laws of organic being. We are no longer puzzled by the origin of language without consciousness or intention, though it is employed with both. We can perceive how it arose out of interjection and mimicry; how a language, symbolizing conceptions and regulated by will, grew out of meaningless sounds resulting from sensori-motor and percepto-motor reflex acts, and gradually assuming an articulate habit. We can grasp the distinction between the mental and the mechanical conditions of speech, and their indissoluble connection with each other. Recognizing the fact that sensory and motor elements are connected together by reflex channels in the ganglionic centres, up to and including the cortex, we are able to dispense with the need, obligatory on Jaccoud and others, of localizing unquestionably reflex phenomena, associated with cerebral disorders of speech, in the infra-cortical ganglia, merely because they are evidently reflex. Conversely, we shall not expect that liberation of the nerves and muscles subservient to speech from the control of the will must entail an outbreak of unbridled reflex actions on the part of the tongue, lips, etc. Inhibitory mechanisms are abundantly present both in and below the cortex, though we are still very ignorant of the distribution of the inhibitory and accelerating energies in the various regions of the brain.[1]

CHAPTER XXI.

Concerning the Functions of the Cortex of the Cerebrum, and their Localization in General.—The Theory of Flourens as to the Functional Equality of the Different Parts of the Cerebrum, and the Law of Substitution in Contrast to Hitzig's Theory of the Functional Difference of the Cortical Convolutions.—Deductions from Veyssière's Discovery touching the Functional Difference of the Anterior and Posterior Cortical Tracts.—The Will as a Sensory Motor Process, and the Act of the Will as a Realized and a Suppressed Movement.—Concerning the Ganglionic Formation of the Key-board of the Will.—Is it at once a Motor-centre and Centre of Co-ordination, or only the latter?—The Ways of Solving this Vexed Question: by Anatomy, by Comparative Anatomy, by Experiment, and by Clinical Observation.—Experiments by Irritation.—Experiments by Mutilation, and the consequent Temporary and Permanent Disturbances of Function.—Explanation of the Restoration of Functions.—Character of the Permanent Derangements of Sensation and Motion after Mutilation of the Cortex.—The Convolutions contain Sensory as well as Motor Apparatuses.

The experience, that sometimes serious losses of the substance of the cortex are not followed by permanent disturbances, is the basis of *Flourens' theory: that all parts of the cerebrum are capable of the same functions, and that each can vicariously*

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[1] Concerning reflex action as the general principle of all movements depending on the nervous system, see *Griesinger*, Ueber psychische Reflexactionen. Archiv. für physiol. Heilk. 1843.—*Laycock*, On the Reflex Actions of the Brain. Brit. and For. Med. Review. January, 1845, and July, 1855.—*Hughlings Jackson*, Clinical and Physiological Researches on the Nervous System. Reprinted from the Lancet, 1875. London, 1875.

perform the functions of every other. The power of portions of the brain to act for other portions Vulpian calls "*Loi de Suppléance.*" We have learned in Chapter XIX. that the radiation of the pes cruris cerebri upward into the corona radiata is divided into sensory and motor districts. Nothing in this justifies us in the assumption that the sensory tracts of the capsula interna can exercise the functions of the motor, or, *vice versâ*, the motor those of the sensory; or that the ganglionic stations, which are situated upon the centripetal track, have the power to forward despatches in a centrifugal direction, or, *vice versâ*, stations upon the centrifugal track centripetal despatches. *Thus the law of substitution undergoes much limitation*, and this alone can be granted, viz., that in the infracortical cerebral regions sensory elements can act for sensory, and motor for motor, and this only "in so far as they find themselves connected in a suitable manner" (Wundt). For, in fact, it is always the connections of the nerve-elements which determine the nature of their functions. This is the chief basis of the localization of the cerebral functions, as of the central functions of the nervous system in general.

It may now be asked whether Flourens' theory is not perhaps correct for at least those parts situated within the limits of the cerebral cortex, within which, indeed, centripetal and centrifugal fibres for all the provinces of the body are closely interlaced. The effort has been made of late, by means of experiments, to gain complete control also over this limited, though yet rather extended field. To have accomplished this is the merit of Hitzig, whose investigations led to new and surprising conclusions, and incited to a great number of fruitful researches. Hitzig arrived at a theory the exact opposite of that of Flourens, viz., *that the cortex is divided into locally distinct sensory and motor tracts. In the anteriorly situated tracts are found circumscribed motor-centres for the different members of the body*, as *e. g.*, for the fore and hind limbs, the jaws and tongue, etc. Only within these regions and centres does he regard it possible for the different nervous elements to act for each other. We cannot enter upon all the details of the lively contention for and against these views, which Hitzig himself at last was compelled to modify; nor can we mention all the interpretations which the new discoveries received; but we must content ourselves with laying stress upon the substantial gain, which in our opinion comes out of it all for the localization of the functions of the cortex, in order to make this available for the localization of the cortical functions of speech.

The well-established fact that the centripetal paths of the pes cruris are directed towards the posterior regions of the cerebral cortex, and the centrifugal towards the anterior, makes it immediately improbable that the different portions of the cerebral cortex exercise similar functions everywhere. It forces us rather to the following assumptions:

a. The sensory impressions, which are carried through the pes cruris to the posterior regions of the cortex, experience at the point where they first enter these parts their earliest cortical modification (Verarbeitung). They scarcely undergo their entire cortical elaboration here, however, but most probably still pass through numerous stations, which possibly extend as far as the anterior regions of the cortex. One should also not forget that it is highly improbable that sensory excitations reach the cerebral cortex only by the paths which traverse the pes cruris. The cortex is also in communication with the thalamus opticus, which is exclusively, or at least chiefly, of a sensory nature. This organ is united to the entire length of the hemisphere by an expanded layer of fibres, without our knowing even at the present day in which regions of the cortex these bundles of fibres are finally distributed.

b. The complicated process of cortical excitation, which we call will, sends out its motor impulses through the anterior regions of the cortex, in which the final cortical elaboration of the proposed movements is accomplished.

As a matter of course, we do not mean by this that the will resides in the frontal cortex, or that this latter has only motor functions. *What we call the will is not only a motor, but always likewise a sensory process.* According to the theory of reflex action, it expends its efforts first in those sensory districts where the impressions from the senses are raised to the level of conceptions of the conscious Ego. The processes of cortical excitation which we call *Reflection* (Ueberlegung) here first attain to the height of a *decision* or *purpose* (Entschluss), and then, endowed with the necessary energy, pass over into the motor districts of the will.

This passing over is not, however, to be regarded as a simple transmission, in the sense that the act of the will—in writing and speaking—is only the translation of thoughts and of the images bound up with thoughts—*e. g.*, of the optical or acoustic word-images denoting thoughts—into a more or less considerable quantity of complicated motor aggregations. *Each act of the will is always likewise the realization of a movement-image previously sketched out in the recollection, or of an entire chain of such movement-images*, which are strung together in part automatically and unconsciously, in part with design and consciously. *Hence, the motor part of an act of the will is also united with sensory processes.*

The question here presents itself, how are we to represent to ourselves that ganglionic mechanism which we will figuratively, but without wishing to convey a false impression, designate as the cortical *key-board of the will*, and which has been styled at one time "*motor centres of the cortex*," at another, "*cortical motor centres of co-ordination*." It is still a mooted question, whether this key-board is to be regarded really as a motor centre, or whether it only excites infracortical motor apparatuses to movement through sensory channels, since it determines, by means of previously registered images, merely the character of the movement, in accordance with the purposes of the conscious intelligence, thus playing only the part of a *centre of co-ordination*.

There can be no doubt that sources of force circulate in the cortex, from which a current develops itself which passes centripetally through the fibres of the pes cruris of the frontal lobe, with sufficient power to set in motion the wheels of the corpora striata, medulla oblongata, and spinal cord; in this sense the cortex has motor power and is a motor centre. It is also well established, that apparatuses exist in the cortex by means of which at the very outset the current is guided to its proper infracortical distribution, as to place and time, and in this sense the cortex is a *centre of co-ordination*. Finally, there is also no doubt that these apparatuses for regulating the details of motor distribution are likewise the same which generate the previously registered movement-images, and hence the motor will-centres represent likewise *sensory apparatuses*. The entire dispute, to state it briefly, revolves around this one point—*Can the cortex rightly be considered as a motor centre also upon anatomical grounds?* Is it provided with any apparatus—such, for example, as the so-called *motor cells*, that are found in the more deeply situated motor centres—not only for regulating the infracortical machinery, for hastening, retarding, or stopping its action, and for attaining this or that form of motion, but also for the generation of living, active force?

The solution of this question is beset with no slight difficulties, because it is impossible in the cortex to distinguish exactly between centripetal and centrifugal channels. Hence, it is not permissible to venture straightway upon conclusions as to the sensory or motor character of collections of ganglia drawn from their situation upon the one or the other track, as one can in the spinal cord, the medulla oblongata, and even higher up in the cerebrum. In the cortex, which represents the uppermost termination and point of rendezvous of the nervous system, all tracks unite in an endlessly intertwisted tissue of numberless plexuses of ganglia, and the determination of the directions which they pursue is perhaps an impossibility.

We can pursue various methods to determine whether motor cells and apparatuses exist within the cortex, which fulfill analogous functions to those of the infracortical motor nerve-cells and apparatuses.

Firstly, anatomy may succeed in finding in the cortex, and especially in those portions of the cortex through which the will sends forth its motor impulses, nerve-cells similarly constructed to those which are found in the more deeply situated ganglia of unmistakably motor character.

Secondly, comparative anatomy should bestir itself to find out whether the development of certain regions of the cortex, and especially of the anterior, stands in a fixed relation to the motor capacities of vertebrates.

Thirdly, we must determine by means of experiment and clinical observation, (*a*) whether the irritation of certain portions of the cortex is followed by movements which do not take place after irritation of other portions of the cortex and brain; and (*b*) whether the absence of certain portions of the cortex is followed by derangements of motion, which can only be explained by the lack of a motor-impelling power.

We shall presently learn what researches in this direction have yielded up to the present time, and will now draw attention to two points only: (1) If it be found from these researches that, as was already rendered probable by previous considerations, sensory functions are consummated everywhere in the cortex, it is not thereby proved that the cortex may not likewise be provided here and there, and especially at the points of emission of the impulses of the will, with especial motor apparatuses. It seems to us that no important consideration stands in the way of the hypothesis, that in those layers of the cortex which emit impulses, both motor and sensory ganglion-cells may exist and may be connected by communicating fibres. (2) Moreover, we will not forget that each act of the will is not only positive, but also suppressed motion. The excitations of the cortex which culminate in an act of the will not only advance downward along the channels of the pes cruris as an impulse, but also in paths still unknown, acting as a check. It is perhaps by the tracks of the tegmentum cruris that these inhibitory currents pass downward.

The following, then, are the most important results of our latest researches into the localization of sensory and motor functions in the cerebral cortex:

1. *Anatomy*.—According to Betz,[1] the surface of the cerebrum is divided by the fissure of Rolando into two halves—an anterior, in which large pyramid-cells, and a posterior, in which layers of nuclei prevail, so that an analogy makes itself manifest with the anterior motor and posterior sensory tracts of the gray cen-

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[1] *Betz* in Kiew, Anatom. Nachweis zweier Gehirncentren. Centralblatt für die med. Wissensch. 1874. S. 578.

tral substance of the spinal cord. The anterior edge of the lobulus quadratus (præcuneus) marks the border of the division on the median surface, as do the fissures of Rolando and Sylvius on the outer surface.

Betz, moreover, discovered still within the anterior region, precisely in situations corresponding to Hitzig's motor centres,[1] in the fourth layer of the cortex, extraordinarily large pyramid-cells lying together in heaps, which he styles *giant pyramids* (Riesenpyramiden); these present themselves nowhere else, and do not reach their full development till after birth.

2. *Comparative anatomy.*—According to von Gudden,[2] the frontal brain is much more strongly developed in the four-footed perpetuum mobile, the squirrel, than in its near relative, the quiet rabbit.

3. *Experiments by excitation.*—a. Numerous experiments first undertaken by Fritsch and Hitzig, and confirmed by others, establish the fact that it is possible in animals to set in motion specified groups of the muscles of the body by galvanic and faradic excitation of specified portions of the frontal region of the cerebral surface—for example, the anterior central convolution, especially in the case of monkeys. The weaker the current one uses, the more circumscribed seem to be these excitable districts; with strong, widely diffused currents, movements can be obtained from neighboring districts lying behind the frontal region (Ferrier). It is not to be supposed, indeed, that the cortex itself is susceptible to electricity (Schiff, Eckhard, Hermann, et al.); it is only the medullary fibres having their origin in the cortex which are irritated. Eckhard succeeded in tracing, by making a series of horizontal slices, a bundle of these excitable medullary fibres (for the anterior extremity) as far as the corpus striatum. It is, however, thereby rendered very probable *that excitations of the cortex find their way from specified points of*

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[1] In man, in the anterior central convolution, and in an especial lobe first named by him, on the median surface, exactly in front of the fissure of Rolando, the lobulus paracentralis. In the dog the giant pyramids lie in the lobe in front of the sulcus cruciatus, which he regards as identical with the anterior central convolution in man and the ape, whilst the central convolutions are said to be wanting in the dog.

[2] Correspondenzblatt f. Schweiz. Aerzte. 1872. No. 4.

the frontal region through specified bundles of fibres to specified groups of muscles.[1]

b. After the discontinuance of the electrical irritation of the so-called motor cortical districts, the movements first obtained repeat themselves everywhere, or only at certain points, as secondary movements, which sometimes result in an epileptiform spasm of the entire muscular system of the body (Fritsch and Hitzig). Eckhard's statement,[2] that it is never possible to evoke such spasms from the posterior surface of the brain, seems to us of great importance, although requiring still further confirmation. This observation reminds one of "*cortical epilepsy*" in man. Circumscribed irritation of the brain cortex, by tumors, cysticerci, inflammatory processes, etc., calls forth at first partial muscular spasms of this or that region of the body, which may gradually increase to general epileptic attacks with unconsciousness.[3] It is still to be settled more exactly, what are the portions of the cortex in man which, when subjected to limited irritation, give rise to spasms, and whether especial partial forms are attached to especial localities.

4. *Experiments by mutilation.*—Mutilation of the cerebral cortex in the rabbit, cat, and dog, produces disturbances in sensation and motion, whether procured by amputation, by circumscribed cauterization—by the admirable method of Nothnagel

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[1] According to Bochefontaine (Gaz. méd. de Paris, 1875. No. 52), the electrical irritation of the centres of Hitzig results also in increased secretion of the submaxillary salivary glands, contraction of the intestines, the bladder, the spleen, expansion of the pupils, etc.

[2] Zeitschrift für Psychiatrie. Bd. 31. Verhandlung des südwestdeutschen Vereins am 2. und 3. Mai, 1874 zu Heppenheim.

[3] Ever since 1861 Hughlings Jackson has frequently, and with emphasis, called attention to the different forms of "unilateral and bilateral discharging cramps," as he calls them. I have myself lately observed two such cases, in which at first circumscribed twitchings of the opposite side, and later universal convulsions, accompanied in the latter case by unconsciousness, were contemporaneous with cortical lesions; there were likewise paralyses of the opposite side. In the one case there was a tumor upon the right parietal lobe; in the other case there was a traumatic hemorrhagic softening of the left third frontal convolution, together with softening of the temporal lobe. In this case there were also present aphasia and hemianæsthesia of the opposite side as far upward as the neck.

Bernhard, Samt, Remak, and Gliky, have published similar observations lately.

in the latter case, or by washing out, by means of a stream of water introduced through holes made by the trepan, the protruding portions of brain-substance, according to Goltz's method.

These disturbances are partly of short duration, in part they last for months—whether for the lifetime is still to be established. These latter we will call permanent, under the supposition that they never entirely disappear. If this supposition be correct, we must attribute these disturbances to the removal of a function absolutely confined to the portion of the brain taken away.

As regards the temporary removal of brain-functions after mutilation of the cortex (or more properly, indeed, in the experiments of Goltz, of one of the hemispheres [Hirnmantel] deep down in the white substance), this condition can be explained partly, though not entirely, by *the law of substitution.* The substitution is possibly a double one: (1) for the portion of the cortex destroyed, another portion in the same or in the opposite hemisphere acts vicariously, this new portion having been already trained to perform the other's functions, or acquiring the necessary degree of training after the injury. One is rather disposed to consider the possibility of substitution through parts of the same hemisphere in cases of slight destruction, and through parts of the opposite hemisphere in very extensive ones. (2) The conducting channels are so constructed that the excitation of separated portions can be accomplished through by-ways (anastomoses between fibres and commissures), so soon as the new path has been sufficiently travelled.

The hypothesis of the substitution of one hemisphere for the other does not suffice for all functions, as Goltz, for instance, has proved.[1] Were this hypothesis true, a symmetrical injury upon the hitherto undisturbed half of the brain would evoke bilateral derangements, when a mutilation had previously been consummated upon the first half and the derangements had subsequently been obviated by substitution. But this does not occur. Goltz mutilated the right half of the cerebrum in two dogs who had recovered from a mutilation of the left cerebrum. Both dogs limped with the left fore foot, revolved towards the right, and fell gently upon the left side, whilst after the first operation all these symptoms appeared upon the opposite side. We must therefore return to the relative autonomy of the infracortical nerve-provinces, in opposition to the cortex which governs them and binds them together as a unit from above. One can conceive that the functions of the infracortical organs are in some way temporarily interfered with by the ablation of parts of the cortex, and that it is not at all, or at least not solely, a question of a loss of cortical functions; the infracortical provinces resume their activity so soon as the inhibiting power diminishes and finally vanishes (Goltz).

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[1] Arch. f. Physiol. 1876. Bd. 13. S. 1.

In respect to the disturbances of sensation, which arise after mutilation of the cortex of the brain, these have been investigated with great exactitude upon dogs by Goltz. They occur both as derangements in touch and in the susceptibility to pain of the skin and as derangements of vision, and according to Goltz it is all one whether the excitable zone of Hitzig or the posterior regions have been injured; the degree of derangement, however, corresponds to the extent of the loss of substance. The same also is said to be true of disturbances of motion, which manifested themselves after extensive mutilations of the posterior as well as of the anterior regions.[1]

The anæsthesia of the skin is lasting, although it can be proved later only by means of the more delicate tests. The same is true for sight. In the beginning, dogs, after a considerable mutilation of one hemisphere, are always blind of the eye of the other side; but the sight returns gradually to this eye, and there remain behind only some very remarkable disturbances of vision, which can be demonstrated only with delicate tests. If we confine those animals from which a large portion of the surface of a hemisphere has been removed, it matters not whether from in front or from behind, to the use of the eye upon the side opposite to the injured hemisphere, the other eye having been destroyed, they are still able to avoid obstacles in their path, but they seem to have lost the perception of depth: they walk over the side of a table; they no longer recognize their food, except by smell, and they are no longer frightened by the sight of things ordinarily causing fear. These derangements cannot be explained by a loss of intelligence, which still exists to a sufficient extent.

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[1] Very diverse from these are the statements of *Ferrier* (Arch. gén. de méd. 1875. p. 503), which he made in the Transactions of the British Med. Association at Edinburgh, 1875, as to the results of his experiments on mutilation in monkeys. He distinguishes between motor and sensory cortical centres. The convolutions bordering on the fissure of Sylvius are, according to him, the seat of lesions in paralyses of the opposite side. The destruction of the frontal regions is without effect upon the movements guided by the will, but it affects the intelligence; that of the under part of the temporo-sphenoidal lobe destroys taste; that of the occipital lobes produces a condition of depression accompanied by refusal of nourishment (!).

Lussana and Lemoigne, who observed the same thing in mutilated pigeons, explained it thus: that after removal of the left eye and cerebrum the still remaining right eye is no longer in correspondence with the still remaining right half of the cerebrum. The animal can no longer, therefore, as they maintain, experience dread at the appearance of threatening forms. On the other hand, they hold that the connection between the right eye and the uninjured mesencephalon and cerebellum remains undisturbed, and the central organs of locomotion, which lie behind the cerebrum, are uninjured, and are properly controlled by the impressions which they receive through the uninjured eye. This explanation seems to be as simple as it is correct in the case of pigeons; but for dogs, Goltz regards it as insufficient, since in these each hemisphere "seems" to be in connection with each eye. He therefore attributes the disturbances to a loss of the sense of color and of place. From these experiments it seems to us that the following conclusion may be drawn: *that in dogs the images which are formed in the subcortical regions of the brain, from the impressions made upon one retina, unite with the representations corresponding to them in the half of the cerebrum on the side opposite to this retina, and thus arrive at conscious perception.* The comprehension of the images is a function of the cerebral cortex.

As for the derangements of motion after mutilation of the cerebral cortex, these are explicable for the most part by deficient co-ordination consequent upon diminished sensation. After inconsiderable losses of substance, one only observes transitory ataxic disturbances. After more extensive injuries, however, there come next paralyses of the opposite side, which soon yield to symptoms of paretic weakness and ataxia (the back of the foot, *e. g.*, touches the floor, instead of the ball of the toes), and to circus movements (Reitbewegungen). Finally, there remains only an entire or partial loss of the ability to use the fore paw for purposes similar to those for which man uses the hand, as in "giving the paw," scratching the head, taking possession of bones, etc. From this one sees that the automatic or machine-like movements, such as running, are restored ; *that the movements acquired by intelligence, however, are permanently lost, or are performed with greater difficulty.*

We do not find that the results of these experiments disprove the presence of motor-cells in the cortex, a thing rendered very probable by the anatomical researches of Betz ; on the other hand, they also fail to prove their existence in this region. While we have learned to regard the cortex as an organ which begets in dogs a certain class of movements, namely those connected with the intelligence, it is still *à priori* probable, from analogy, *that to it belong also those special anatomical apparatuses which are present in all other motor centres, and which, as is supposed, in the form of so-called motor cells, serve to change latent elastic forces (Spannkraefte) into actual mechanical power, producing the contraction of muscles and the raising of weights.* Still the ganglionic arrangement of the cortex exhibits much that is peculiar in contrast to other centres ; motor cell groups occupying entire convolutions, and corresponding in any degree to the motor nuclei of the oblongata and the gray anterior columns of the spinal cord, do not exist in the cortex ; in this sense there are no purely motor convolutions. *Upon the one hand, the chiefly motor convolutions or centres of Hitzig seem to interpose in sensory processes, whilst on the other a portion of actual power, which, in the form of will, turns the motor wheels, probably springs from the sensory districts of the cortex.*

It has been very properly pointed out that the derangements of motion, after ablation of the cerebral cortex in animals, call to mind the ataxias and paralyses which are observed in the progressive paralysis from cortical atrophy in man. Meynert has also repeatedly laid stress upon the fact that, after paralytic insanity with delirium, atrophy of the frontal lobes is the chief lesion. It has, moreover, been long known that extensive lesions of the cerebral cortex result in paralyses of the opposite side, sometimes of a temporary, sometimes of a permanent character, and it seems that it is rather rapidly developed lesions, as, *e. g.*, necrotic and hemorrhagic softenings of the posterior frontal and anterior central convolutions, which produce such paralyses. This is, however, all which up to the present moment we are at liberty to affirm in regard to the localization of the cortical functions in man, and with the manifold difficulties and sources of error which here come into play, it is impossible as yet to postulate anything more definite as to the functional capacities of individual cortical regions.[1] So far, the greatest advances have been made towards an understanding of the relations in which the function of speech stands towards the divisions of the cortex, and upon the consideration of this we will now enter more closely.

CHAPTER XXII.

The Guiding Principles for the Localization of the Cortical Functions of Speech, and the Nature of these Functions.—Experiments touching the Cortical Centre of Movements of the Mouth.—Clinical Difficulties in the Localization of the Functions of Speech in the Cortex.—Necessity of the Law of Substitution for the Explanation of Clinical Experiences as to the Results of Circumscribed Removal of Cortical Substance.

Now that we enter upon the much discussed problem of the localization of the cortical functions of speech, let us take for the solution of this task the guiding principles from the results mentioned in the preceding chapter.

Our previous researches have shown what are the functions of speech belonging to the cerebral cortex. We found that only the apparatuses for the mechanical consummation and connection of the movements of articulate phonation exist in the infracortical regions of the brain; that the formation of syllables and words, however, as is required for the purposes of speech, takes place in the cortex. Articulation, in so far as it is an intellectual task, must be regarded as a cortical function. The infracortical apparatuses for articulation simply accomplish the formation and union of sounds in that strength, rapidity, and sequence in which the cortical sound-keys are struck. We may further assume that the perception of articulate sounds simply as phenomena of noise, or of text-characters as simple optical phenomena, takes place in the infracortical regions; whether, however, their recognition as acoustic or optical images of this or that characteristic form is completed underneath the cortex, is uncertain; in any case, though, their comprehension, that is, their union with the necessary conceptions, their use as signs for the expression of the thoughts, takes place in the cortex. Here also take place the processes of excitation, by means of which a word as an acoustic or optical image is transferred in its sensory sound-divisions to the sound key-board (Lautclaviatur), where the word-text, previously transposed into the note-text of remembered movement-images, is played off. Finally, the cortex is the secret workshop of the thoughts; here the representations, as they develop themselves out of the divers sensory and motor operations of the nervous system, are conceived, joined together in logical membership, and by means of especial associatory processes translated into grammatically shaped and syntactically constructed word-symbols, which then, through the key-board, reach motor expression.

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[1] Compare *Samt*, Concerning ataxic and paralytic derangements of motion in progressive paralysis. Arch. f. Psychiatrie. Bd. 5. S. 112. f.—*Meynert*, Vierteljahrschr. f. Psychiatrie. 1867. S. 166, und Arch. f. Psychiatrie. Bd. 4. S. 417.—*Bernhardt*, Arch. für Psychiatrie. Bd. 4. S. 698.—*Samt*, Zur Pathologie der Rinde. Ibidem. Bd. 5. S. 201.

It is almost superfluous to remark here again, that we must count as a part of the cortical division, which has to perform these important services, not only the several layers of gray substance on the surface of the hemispheres, but also the massive fibræ arcuatæ in the white medullary substance of the hemispheres, which, as Meynert's Association-System, unite together the cortical convolutions of different regions. The fibrous system of the corpus callosum also, which unites the cortical convolutions of both hemispheres, is to be added to this department.

We will not enter with high soaring expectations upon the giant task of tracing up to this or that portion of the cortex, the different functions of speech of the cerebral cortex, since we have discovered how modest is the result which previous scientific investigation of the functions of the cortex has in general attained in this direction. We will especially turn away with a smile from all the naïve attempts to locate a "seat of speech" in this or that convolution of the brain. It is à priori probable that an enormous association-tract in the cortex has been assigned to speech, even though the sound key-board may be confined to the anterior cortical regions, through which the impulses of will have their outlet, since it (speech) must be bound up with the entire tract of conscious conceptions, and this indeed stretches across the entire cortical region. Probably the entire cortex takes part in thought, although the individual conceptions must be realized through the intervention of different plexuses of cells, according to the sensory sources out of which they are formed. We must not, indeed, so regard this localization as if one conception were bound up with one particular point, and another with another, or as if one cell served only for this and for no other conception. We may assume that the functional connections, whose excitations are transposed into conceptions of this or that class, extend over wide regions. Moreover, the simplest abstraction is bound up with numerous intuitions, movements, and judgments of the senses. It is further very probable that the same cell can enter into very different connections, and aid in the construction of different conceptions. The cortex, too, is the very organ in which, after an injury to its substance, the endless interlacing of the channels contained in it guarantees to the law of substitution an extensive, though not unlimited application.

For all those who do not hold the view that words and thoughts originate from sources above and outside the nerve substance, the localization of the functions of speech in portions of the cortex follows as a necessary postulate of logic; the reason for this lying in the fact that, after circumscribed injuries of the cortex, sometimes only the motor word with its image of movement, sometimes only the sensory word as a representation of articulate sound or of writing is wanting, and sometimes only the connection between a word and its conception is interrupted. The motor-formation of words must reach completion by other channels than those of the acoustic and optical word-images, and these by still others than those of conception. But so soon as we attempt to designate more exactly these paths by the aid of clinical experience, we are brought up against difficulties which are too great for us. We are speedily led to the conclusion that the paths of speech are so intertwined with each other and with those of conception, that we cannot succeed in disentangling them and marking down the separate stations of the labyrinthine way. The region of the key-board of speech only can be pretty exactly ascertained.

Physiological experiment, as we might expect, leaves us here in the lurch ; we are thrown entirely upon clinical observation. One solitary result of the experiments of Hitzig and Ferrier with electrical irritation is of significance for our problem. These investigators assure us that it is possible to evoke movements of the mouth, tongue, and jaws in dogs and monkeys, by placing the electrodes on the under part of the frontal lobe, at the point where it borders on the fissure of Sylvius. In the more precise designation of the place, they differ somewhat from each other, it is true. Hitzig locates his motor cortical centre for the above-mentioned muscular regions in the monkey in the lowest part of the anterior central convolution, whilst Ferrier places the same close in front of this, in a region corresponding rather to the so-called tract of Broca in man. Hitzig found, likewise, that from this point he obtained not only unilateral movements of the regions of the mouth on the opposite side, but also bilateral. It appears, therefore, that the fibres of the cortex leading to the muscles of the region of the mouth proceed from the designated cortical tract, and that the excitation of one hemisphere suffices to throw into contraction the muscles of the mouth. The results of this experiment coincide with a fact, to which we shall recur more in detail farther on, viz., that in man, in the great majority of instances, it is the lesions of the frontal and insular region, and especially of the posterior portion of the third frontal convolution bordering on the lower part of the fissure of Sylvius, the region of Broca, by which speech is much interfered with, and that lesions of one side alone suffice to produce such effects.

The difficulties which stand in the way of the localization of the functions of speech in the cortex, by the aid of clinical observation, are of several kinds.

1. *The coarse anatomy of the cortex is a science of very recent date.* All of the older observations are scarcely available as regards anatomical points, and are generally very inexact.

The researches of Gratiolet (1854) first clearly established the relations as to form of the surface of the human cerebrum, and made possible a more exact topographical description of its lesions, as was first carried into execution by Broca (1861). The useful little monograph of Ecker[1] makes it easy to-day for any one to accurately designate the position and extent of cortical injuries.

In order to understand how superficially localizations are recorded even in the writings of the best of the older observers, let one cast a glance at the principal works of such men as Bouillaud, Lallemand, and Rostau, to whom, notwithstanding, the pathology of the brain owes so much. They are content to indicate the affected lobe with the general designation, "in front," "behind," "in the middle," etc., and with an equally indefinite appreciation of the extent of the lesion. One seldom learns whether the cortex or the white medulla, the corpus striatum, centrum semiovale, etc., was the seat of the lesion. Andral and Durand-Fardel are indeed more exact, although they seldom reach our present topographical requirements, and never in the cortex. Even so great an anatomical authority as Cruveilhier gives an incorrect description of an injured brain, as a reference to his accompanying admirable plate shows (compare Chap. XXIII.). Laborde[2] remarks, in a work published in 1866, that, according to his belief, his predecessors had overlooked the frequent simultaneous occurrence of points of softening in the cortex and corpus striatum, only because in examining the brain they had made too hasty and too few sections; but his own specifications of locality are equally unsatisfactory.

An anecdote which Broca[3] relates is very instructive. One day Duchenne informed him that Trousseau had found the region of Broca uninjured in a man who suffered with aphasia. Immediately Broca repaired to the Hôtel-Dieu, and discovered that Trousseau had overlooked a softening of this tract, which existed near one of the parietal and island convolutions. When a man so conscientious as Trousseau allowed himself to be guilty of such an oversight, in the midst of the dispute over Broca's hypothesis, and after Broca had insisted with the utmost vigor

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[1] Die Hirnwindungen des Menschen u. s. w. Braunschweig, 1869.

[2] Le ramollissement et la congestion du cerveau, etc. Paris, 1866.

[3] Bulletin de la soc. d'anthropologie. 1863. p. 201.

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upon the necessity for an exact determination of locality, what are we to expect from the great mass of ordinary observers?

The records of cases which Bateman collected together from almost the entire literature of educated countries, in his monograph "On Aphasia," published in 1869, shows in an astounding manner how badly the material bearing upon disturbances of speech had been dealt with up to that time, from an anatomical, and, unfortunately, also from a clinical point of view.

2. Just as little can critics profess to be content with the description of derangements of speech by most of the older and many of the more recent observers. If anywhere, then here should obtain the saying of Morgagni, that one should weigh and not count cases.

In the older observations, stammering and stuttering are not once distinguished (compare Chap. XXXIV.). Duchenne first sketched out a more exact delineation of disturbances of speech in bulbar paralysis (1860). A year later, 1861, Broca first characterized aphasic disturbances of the cortex in two admirably described cases, for which he proposed the term "Aphemia," which was exchanged by Trousseau for the now commonly used "Aphasia."[1] Trousseau afterwards distinguished more exactly between aphasia and other forms of deranged speech. Leyden, in 1867, first contrasted aphasia with anarthria. William Ogle, in the same year, made a distinction between ataxic and amnesic aphasia. Steinthal (1871) separated the syntactical derangements of speech, under the name of akataphasia, from these other two forms of deranged word-formation. How carelessly one rushed at the diagnosis of aphasia, and that not long ago, appears from a remark of John Ogle,[2] that some one had brought a patient to him as aphasic who spoke indistinctly on account of an absence of the tongue!

3. It is by no means always easy to carefully establish, according to symptoms, the category of any given affection of speech, and to ascertain always what form of dumbness one has before one, whether one of difficult and altered speech, or of impeded comprehension of speech.

What difficulties may we not have simply to establish whether a man cannot or will not speak? Why an individual does not attend to conversation? Whether he does not hear it or will not hear it? Whether he does not understand the words on account of weakened intelligence, or on account of absence of mind, or because he no longer recognizes the word-images? Whether he mutilates the words, and, stammering, distorts the sounds on account of bad training, on account of congenital defective development or paralysis of the tongue, and the like? We have already previously demonstrated how difficult it often is to be sure of the milder forms of paresis of the tongue, lips, and palate?

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[1] According to the suggestion of the learned Greek scholar, Krisaphis, made up of ἀ privative, and φάσις, speech.

[2] Lancet. 1868. March 21.

4. Under such circumstances, one can readily appreciate how little aid we shall derive, in our task, from using the statistical method. Single observations can outweigh whole tables, partly on account of their extreme exactness, and partly on account of their inherent force of demonstration. This inherent worth is shown by different factors. The more sharply a lesion is confined to one spot, the more quickly and completely also does the structure of the affected spot become injured thereby; the healthier the individual was previously, and the more sudden, the more marked, the more lasting the subsequent derangement of speech, with just so much greater certainty may we deduce the disturbance from the lesion of the affected spot

There are many persons, and especially the debilitated and nervous, who require but a slight disturbing force to render them speechless for a shorter, and, indeed, in some instances, for a longer time. An excitement of the feelings, a moral shock, a rush of blood to the head, a rapidly-developed ischæmia of the brain, such as easily occur in affections of the heart and scleroses of the arteries, suffice to bring about in such people dysarthric, dysphasic, or dyslogic disturbances. Should a circumscribed coarser lesion be present at the same time in such a case, one might readily commit the error of ascribing to it the disturbance of speech, although this perhaps originated in the congestion or ischæmia of other parts.

5. Those factors which establish the intrinsic value of a case are met with, in the different morbid processes which lead to the destruction of portions of the cortex, in very unequal ratio. Referring to the previously mentioned (p. 684) considerations on this point, we will now only remark that by far the most valuable material for clinical study is afforded: (1) by *necrotic softening*, especially as a result of the closure of small terminal arteries by embolism or thrombosis; (2) by *injuries* which penetrate only the substance of the cortex, the direction and extent of which can be exactly determined. These latter are especially adapted to elucidate the immediate consequences of a circumscribed interruption and irritation of the cortical tracks. Points of softening afford the best opportunity of ascertaining the more

permanent sequences of the removal of a portion of the cortex. *Hemorrhagic foci* and *abscesses* stand in the second rank. For reasons already stated, observations on tumors and scleroses of the cortex are to be used with the greatest caution.

6. *The symptoms* which follow, either temporarily or permanently, a lesion of the cortex *are at times phenomena of stimulation, at times those of paralysis.*

In regard to *the phenomena of stimulation*, we must not forget that they can be evoked by irradiation and reflex action from distant organs, with which the portion of the cortex is connected; their local interpretation is therefore always difficult. This is true, *e. g.*, for the phenomenon of stuttering, which has been observed, among other things, in the limited encephalitis of the posterior districts of the cortex. It does not show that here, posteriorly, motor districts were directly involved in the disease.

If a function is only temporarily wanting, in cases of *paralysis*, we are still not justified in ascribing this function to the injured or destroyed portion of the cortex, since its absence may also possibly be explained as an inhibitory phenomenon due to irritation, in the sense used by Goltz. This comes into consideration, namely, in cases of injury to the brain and inflammatory deposits; less so in the necroses due to thrombosis. The permanent cutting off or disturbance of a function is, on the other hand, to be referred with certainty to the portion of the cortex abstracted. If we see the same derangement of function, after destruction of a portion of the cortex, result in one case as a temporary thing, in another permanently, we may then assume that this part is, indeed, entrusted in some degree with the affected function; that, however, an accommodation could only be brought about in the last, and not in the first case, by means of substitution. Without this hypothesis it is incomprehensible why lesions of one and the same cortical division are followed, perhaps in three consecutive cases, by permanent disturbances of function of the same nature, and then in four or five cases by only temporary ones. Since it is not possible to explain dysphasic disturbances by a resumption of the function of infracortical organs, as is permissible for certain locomotor functions, there remains no other resource for us but the supposition that in these cases the vicarious activity takes place through preserved portions of the cortex, either in districts of the same hemisphere with the requisite connections, or in symmetrical regions of the other hemisphere.

It was noticed even by Broca that the extent of the lesion of a convolution stands in no fixed relation to the extent of the consequent aphasic disturbance. We also sometimes see inconsiderable mutilations of a certain tract injure a given function permanently, and, again, for only a short time. The reason of this is still entirely obscure. There are, perhaps, in every district especially important points of union of the cortical paths for which a substitute is more difficult than for other points. Great individual differences also exist in the anatomical disposition of the cortical paths, as Flechsig proved for the paths in the spinal cord.

CHAPTER XXIII.

Literature of the Localization of Speech.—Gall.—Bouillaud.—Marc Dax.—Broca. —Formularization of the Questions to be asked at this Point.—Does the Entire Destruction of the Two Anterior Lobes cause Loss of Speech?—Do Aphasic Disturbances originate only in Lesions of the Anterior Lobes, and especially of the Anterior Hemispheres proper?—Do Aphasic Derangements occur only, or even principally, in Lesions of the Left Cerebral Hemisphere?—Do Lesions of the Left Anterior Lobe lead more frequently to Aphasic and Mingled Anarthric and Aphasic Disturbances than those of other Portions of the Brain?—What Region of the Left Anterior Lobe do we find oftenest Injured in Aphasic Derangements?—Does the unusually frequent Concurrence of such Derangements with a Lesion of the Left Island Region bear any Relation to the great Frequency of Left-sided Embolic Softenings?—Is the Third Frontal Convolution of Vital Importance for Speech?—What is the Basis of the Pre-eminence of the Left over the Right Hemisphere in Speech?—Broca's Theory.—Causes of the Predominating Use of the Right Hand.—Can one distinguish, within the Cortical Region of Speech, Centres for the Motor Co-ordination of Words and Acoustic Word-Images?

The literature of the localization of speech is associated mainly with the names of Gall, Bouillaud, Marc Dax, and Broca.

Gall is the proper father of this idea. He distinguished between "*word-sense*" and "*speech-sense*," and referred these capacities to the frontal lobes behind and above the orbits.

His pupil, Bouillaud, deduced speech from two elementary processes connected with the brain: the formation of the internal words as symbols of thought with the memory of words, and the formation of external words. The latter, he maintained, is governed by an especial co-ordinating principle. This "*principe législateur de la parole*" he located in the anterior lobes of the brain, *i. e.*, in the divisions of the cerebrum over the fissure of Sylvius and in front of that of Rolando.[1]

Marc Dax recognized the strikingly frequent concurrence of derangements of speech with lesions of the left cerebral hemisphere.

Broca confirmed this, and referred the remarkable fact to the same causes which make most men right-handed, viz., that they preferably use the left cerebral hemisphere in the more delicate tasks. At the same time he ascribed an important rôle to the third frontal convolution, and especially to the posterior third of the same, in the power of articulate speech. Most people—all right-handed people, exercise only the left third frontal convolution in speech.

Gall has the great merit of having introduced the method of dissecting the brain from below upward, of having recognized the continuous medullary strands of the brain, and of having traced them from the spinal cord below up to their radiation in the cerebral cortex. This furnished, therefore, the necessary anatomical foundation for the localization of brain-functions: the connection, namely, of the ganglionic central masses of the brain, by means of isolated bands of fibres, on the one side with the senses, and on the other, with the movable members of the body![2] The *word-sense* is, according to Gall, merely an especial memory for words, which is independent of the intelligence, as the case of that child proves, who at the age of five years learned by heart all the fables of Lafontaine, and afterwards still a whole volume of the "Cours de Mathématique" of Bezout.

Different from this, according to Gall, is the *speech-sense*, the *philological talent*, the ability to investigate the genius of languages. In the preface to his great work he relates in detail how he was struck, even as a school-boy, with the varying mental gifts of his fellow-scholars, and especially with the concurrence of large staring eyes (goggle-eyes) with the ability to commit things to memory. By this observation he was conducted, as he declares, to his brain-studies—which form an epoch—and, unfortunately, also to his great errors in phrenology and cranioscopy. Goggle-eyes, he declared, indicate word-sense, "Schlappaugen"—that is, downward projecting eyes (yeux pochetés), speech-sense.

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[1] That is, not merely in the cortex! *Trousseau* reckons, among the deeper portions of the brain, the anterior half of the island and the corpus striatum as a part of the anterior lobe.

[2] *Gall* et *Spurzheim*, Anatomie et physiologie du sytème nerveux. Vol. I.-IV. Paris, 1810–1819.

Thomas Hood published in the Phrenological Transactions, Vol. III., of the year 1822, the first case of aphasia more carefully described and illustrated by an autopsy; there was a lesion of the left frontal lobe.

Bouillaud's doctrines have been presented in his well-known work upon cerebral inflammation,[1] in an especial treatise (1825),[2] and in several very lively and often hotly contested debates before the Paris Academy of Medicine.[3] He also made experiments upon animals as to the functions of the cerebrum, in order to support his statements.[4] He collected gradually more than five hundred cases of his own and others, very few only of which, however, respond to critical demands. Bouillaud properly distinguishes the movements of the tongue as an instrument of speech from its movements for other purposes of the will, and the speech paralysis of the tongue from its other forms of paralysis. Bouillaud did not succeed in gaining the assent of a Lallemand, Andral, Cruveilhier, Trousseau, and other celebrated contemporaries, to the localization of his "principe législateur de la parole" in the frontal lobes. The chief objection which was made to it was that the power of speech had been preserved after supposed total destruction of both anterior lobes. On that account Bouillaud offered a prize of 500 frcs. for whoever should authenticate such a case. He never would acknowledge that it had ever been observed.

Marc Dax, a physician of Sommières (Gard), delivered (1836) to the meeting of physicians at Montpellier an essay, which, in spite of its surprising contents, long remained unnoticed.[5]

He had, in every case of hemiplegia with implication of speech, since 1800, found the paralysis always on the right side and the cerebral lesion on the left; if the lesion existed on the right side, then speech was not affected. He collected a quantity of observations from the literature of the subject, which seemed to him to establish the coincidence as a law. This treatise first excited attention when G. Dax, the son, laid before the Academy in 1863 an essay,[1] in which—referring to his father's paper, and to 140 cases, most of which occurred in the practice of others—he postulated the constant concurrence of disturbances of speech with lesions of the left hemisphere. The reporter Lelut disposed of the subject summarily with a few contemptuously discouraging words, and declared all attempts to localize speech to be simply "Phrenology," which he had previously branded in two brochures as a "pseudo-science."

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[1] *Bouillaud*, Traité de l'encéphalite. Paris, 1825. p. 157 seq.

[2] Recherches cliniques propres à démontrer que la perte de la parole correspond à la lésion des lobules antérieurs du cerveau, et à confirmer l'opinion de Mr. Gall sur le siège de l'organe du langage articulé. Arch. de Méd. 1825.

[3] Bulletin de l'Acad. de Méd. 1839. T. IV. p. 282-328. Ibidem. 1848. T. XIII. p. 699-779. Ibidem. 1864-65. T. XXX. p. 575 seq.

[4] *Magendie*, Journ. de physiol. expér. et pathol. T. X. 1830. p. 36.

[5] *M. Dax*, Lésions de la moitié gauche de l'encéphale, coincidant avec l'oubli des signes de la pensée. 1836. Published in the Gaz. Hebdom. 1865, Apr. No. 17.

This gave Bouillaud occasion to enter the lists, not for the support of the exaggerated and therefore false statement of G. Dax, but for the localization of the power of speech in general, which led to one of the longest and most important discussions ever held upon this subject.

Broca, previously an opponent of Bouillaud's localization of the power of articulate speech, came forward (1861[2]) with a dogma which excited universal attention, and which adopted the hypothesis of Bouillaud and marked its outlines more precisely: "The integrity of the left third frontal convolution, and perhaps also of the second, is essential for the development of the power of articulate speech." His proposition was supported by two cases, both carefully observed, and minutely analyzed.

In the next two years the Paris doctors, at the autopsies of all aphasic persons, to which Broca was invited,[3] out of fifteen cases found fourteen times lesions causing destruction in the posterior third of the left third frontal convolution. In all these cases the lesion was not confined to this region; there were always still other neighboring portions simultaneously affected, sometimes these, sometimes those, and in different degrees; but as to the one point in question, they all coincided. They seemed, therefore, to render it necessary that we should regard the posterior third of the left third frontal convolution, which has since been designated as *the region of Broca*, as the portion of this convolution essential to articulation. Only in one case (Charcot's[4]), Broca found nothing in the left third frontal convolution except an unimportant alteration in the form of a fatty degeneration of the capillaries, whilst the left temporal lobe and the left island were extensively destroyed by masses of softening.

On top of this came an observation of Parrot's,[5] which seemed especially suited to place in a clear light the great significance of the left third frontal convolution contrasted with the right. In the case of a woman, afflicted since childhood with a contraction of the left arm, whose speech was unchanged, the right third frontal convolution with the island was found destroyed.

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[1] Observations tendant à prouver la coïncidence constante des dérangements de la parole avec une lésion de l'hémisphère gauche du cerveau. Bull. de l'acad. de méd. 1864-65. T. XXX. p. 173.

[2] Sur le siège de la faculté du langage articulé, avec deux observations d'aphémie (perte de la parole). Bull. de la soc. anat. T. VI. Août, 1861.

[3] Remarques sur le siège, le diagnostic, et la nature de l'aphémie. Bull. de la soc. anat. Juillet, 1863.

[4] Gaz. hebdom. 1863. p. 473.

[5] *Broca*, l. c. Juillet, 1863.

Next,[1] Broca proposed a hypothesis for the explanation of this superiority of the left hemisphere; he attributed it to the predominating use of the right hand by the majority of people, and on this basis explained the cases in which, with congenital absence of the left third frontal convolution, speech was still acquired. Such a case, observed by Moreau (in Tours), was communicated by himself. A person forty-seven years of age, from earliest childhood an epileptic, learned to speak, to read, and to sew with the left hand, although the entire left first primitive convolution which surrounds the fissure of Sylvius was wanting. She knew how to express her ideas very well. This person was forced to exercise the right cerebral hemisphere in sewing and speaking, because the left was not available therefor on account of the absence of the first primitive convolution. The ability to learn to speak is therefore, according to Broca, dependent upon the integrity of one at least of the third frontal convolutions. Most people exercise only the left third frontal convolution for this purpose.

The questions which were debated by Bouillaud, Dax, and Broca, we will now strive to formulate separately and to answer.

1. *Does the entire destruction of both anterior lobes entail the loss of the power of speech?*

The error of Bouillaud's views would be immediately proved, did observations exist in which both the anterior lobes[2] have been entirely destroyed without loss of speech. A well established case of this kind would be positive proof that speech is not limited to the anterior lobes. By such a case Broca's theory would likewise be upset, according to which articulate speech is only possible where at least one of the two third convolutions of the brain remains uninjured.

So far as we have succeeded in searching the literature, there is no trustworthy observation where one has found with unaffected speech a total destruction of both anterior lobes, or even only of the collective cortical tracts in front of the fissure of Rolando and that of Sylvius. Those alleged cases of this kind which have been cited against Bouillaud by very well-recognized authorities, do not bear criticism. In this we must do justice to Bouillaud.

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[1] Du siège de la faculté du langage articulé dans l'hémisphère gauche du cerveau. Bull. de la soc. d'anthropol. T. VI. 1865. Juin 15.

[2] We use this designation always in the sense of *Bouillaud*, according to *Trousseau's* commentary.

This is true, namely, of the often quoted case of Cruveilhier,[1] which we considered in the last chapter. An idiot, twelve years of age, gave expression to her longings for food by clearly articulated words. According to the text describing the case, both anterior lobes were entirely wanting; according to the drawing, considerable portions of both lobes were preserved, and especially the larger portion of the left third frontal convolution.

In order to draw out Bouillaud, Velpeau[2] demanded the prize of 500 francs for a case in which a lobulated cancer had professedly destroyed the anterior lobes; in truth, however, it had left untouched the greater part of them, and especially two-thirds of the left frontal lobe. The subject, a hair-frizzler, sixty-six years of age, had constantly indulged in obscenity up to the last days of his life.

On the other hand, it distinctly appears from cases just mentioned and from others since published, that sometimes large portions of one or both frontal lobes are damaged, without articulate speech being on that account destroyed. Among all of these cases, however, there is not one, so far as we have been able to discover, in which the third frontal convolutions on both sides were wanting. The cases of mutilation of both frontal lobes observed up to the present time testify distinctly, therefore, neither against the theory of Bouillaud, nor against that of Broca.

Bergmann[3] has communicated a new and very remarkable case of extensive traumatic injury of both frontal lobes without loss of speech; still here also the upper part of the left frontal lobe, together with the frontal convolutions of the right side, remained intact.

2. *Do aphasic disturbances result only from lesions of the anterior lobes, and especially of the parts of the hemispheres proper situated in front of the fissure of Rolando and that of Sylvius?*

To this the answer is to be decidedly given, that, as well in temporary as in permanent aphasic disturbances, the anterior lobes have not seldom been found intact and other regions of the cerebrum destroyed. For instance, aphasia has been observed in lesions of the island lobe, of the parietal and temporal lobes, or of several of these at the same time. We shall become acquainted with examples enough of this nature.

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[1] Anatomie pathol. VIII. livr. pl. 6.

[2] Bull. de l'acad. de méd. 1843. p. 862.

[3] Virchow's Jahresber. für 1872. Bd. 2. S. 52. The observation of *Avonde* is of a similar nature, but it excites very little confidence. (Thèse de Paris, 1866.)

3. *Do aphasic disturbances occur only, or even chiefly, with lesions of the left cerebral hemisphere?*

We have previously seen that anarthric disturbances of the severest character proceed from the oblongata, the pons, and the tract of the intra-hemispherical channels of the will (including the corpora striata); nevertheless, as regards the latter, it has been made plain that lesions of the left hemisphere interfere with articulation much more seriously than do those of the right.

If one pays regard to the aphasic and mixed aphasic and anarthric derangements with and without hemiplegia, equally whether they proceed from injuries of the cortex alone, or from such as involve both this and the caudex cerebri, their strikingly much greater frequency and permanency of occurrence in lesions of the left hemisphere must be accepted as an established fact. Seguin,[1] in New York, out of 260 cases of hemiplegia with concurrent aphasia, calculated the proportion of the aphasias in consequence of left-sided hemispherical lesions to those in consequence of right-sided ones, to be as 243 : 17, that is, as 14.3 : 1.

The ground for this remarkable difference has been sought in the fact that, apart from the question of aphasia, the left hemisphere is more frequently the seat of lesions than the right. This is, however, not correct, since, according to the investigations which Vulpian and Charcot made at the Salpêtrière, one finds both hemispheres about equally often affected. For 58 hemiplegias from lesions of the right hemisphere, they recorded 52 from lesions of the left.

4. *Do lesions of the left anterior lobe lead to aphasic derangements oftener than those of other parts of the brain?* To this question we must also answer unhesitatingly, *Yes.*

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[1] Quarterly Journ. of Psycholog. Medicine, January, 1868.—In *Hammond*, A Treatise on Diseases of the Nervous System. New York, 1872. p. 199.

It has been sought to establish statistically the proportion of aphasias in lesions of the left anterior lobe (verified by autopsies) to those in lesions of other portions of the brain. Seguin placed it at 514 : 31. But the tables, which he collected mainly from the writings of the two Daxes and of Bouillaud, do not stand criticism. What shall we say to this, not to speak of anything else, that these authors have recorded 466 cases of aphasia, all with lesions of the left anterior lobe, and not a single one with lesions of other parts of the brain?

Somewhat more available is the testimony of Voisin,[1] who in aphasia found the left anterior lobe affected 140 times, the right anterior lobe only 6 times. As for the rest, he speaks of aphasias from lesions in the oblongata and pons, which shows that he did not know how to distinguish at that time between anarthria and aphasia.

The most instructive report on this question seems to us to be that published by Callender.[2] He compared, with reference to this very point, a great number of lesions of the brain accompanied by hemiplegia, as they presented themselves to him and to Kirkes in autopsies, one after another, in the course of a series of years at St. Bartholomew's Hospital, in London. Deducting two cases of coma with right, and four cases with left hemiplegia, in which the disturbances of speech could not be determined, out of thirteen cases of right hemiplegia, aphasia was present twelve times, and only once absent, in which case a hemorrhagic clot lay farther behind, that is, under the end of the lateral horn of the lateral ventricle. In thirteen cases of left hemiplegia, on the other hand, genuine aphasia did not occur once; but in four cases there was a slight indistinctness of speech from paralysis of the muscles. To this are to be added, moreover, three cases of preservation of speech with lesions of the left half of the brain, in the posterior regions.

5. *Which portion of the left anterior lobe does one find most often affected in aphasic derangements?*

The experiences of all physicians agree, with singular unanimity, that it is especially the region of the island, with the neighboring portions of the frontal lobe and corpus striatum, injuries of which give rise to aphasic and mixed anarthric and aphasic derangements.

Lohmeyer[3] collected 53 carefully described cases of aphasia, in which an autopsy had been made. In 50 cases the aphasia depended upon a lesion of the left hemisphere, and among these the lesion was 24 times in the third frontal convolution; 34 times in it and its neighboring parts, including the island, which was 7 times concurrently affected; 13 times in the left island; 6 times in this alone; only twice did it follow lesions of the anterior portion of the frontal lobe, thrice of the middle lobe at the fissure of Sylvius, twice of the middle and posterior lobes, four times of the posterior lobe.

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[1] Art. Aphasie, Dict. de méd. et de chir. prat.

[2] St. Bartholom. Hosp. Reports. London. III. p. 415. 1867. u. V. p. 3. 1869.

[3] Archiv f. klin. Chirurgie, 1872. XIII. S. 309.

Among the lesions leading to aphasia, necrotic softenings, from embolism and thrombosis of the artery of the fissure of Sylvius, play a principal, even though by no means an exclusive part. Hemorrhages, abscesses, and tumors of this region produce the same effect. Jaccoud, in recalling the fact that cerebral softenings from embolism occur much more frequently on the left than on the right side, and that these take their origin mainly in the artery of the fissure of Sylvius, which supplies this region with blood, is disposed to explain the more frequent occurrence of derangements of speech in lesions of the left hemisphere simply by the greater frequency of embolism of the left artery of the fissure of Sylvius. Statistics, however, do not justify such an assumption.

It is, indeed, correct that in the two tables of Meissner,[1] out of 38 cases of one-sided embolism of the artery of the fissure of Sylvius or of the carotid, these arteries were plugged 26 times on the left and only 12 times on the right side. Bertin[2] even found embolism of the left carotid, cerebral artery, and the artery of the fissure of Sylvius, 31 times, and on the right side only 7 times. But, if we compare at the same time the proportion of the foci of degeneration in the brain, upon the right side and the left in general, without reference to their nature, we shall find that they occur rather more frequently on the right side than on the left. In 169 cases, Andral[3] found these lesions 73 times on the right side only, 63 times on the left side only, 33 times in both hemispheres. Softenings form certainly a very large proportion of the degenerations of the cerebrum which produce aphasia; but, in addition to them, hemorrhages, abscesses, and tumors occur as frequent causes of aphasic disturbances.

If we take the proportion (as 31 : 7) of lesions from embolism of the left hemisphere to those of the right, from the tables of Bertin, which are the most unfavorable to the right side, and compare with this the previously given proportion of aphasias accompanied by right hemiplegia to those with left hemiplegia, which Séguin found to be as 243 : 17, then we have in the first case 4.4 : 1 only, in the second 14.3 : 1.

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[1] *Schmidt's* Jahrb. Bd. 117. S. 248. Bd. 131. S. 340.

[2] Ibidem. Bd. 147. S. 288.

[3] Clin. med. III. édit. J. v. pp. 377, 378.

From this it is perfectly certain, *not only that the lesions of the left hemisphere are much more frequently connected with aphasic and mixed anarthric and aphasic derangements than those of the right, but also that it is chiefly the anterior portions of the left hemisphere, and, from amongst these, especially the portions bordering on the region of the island, which lead to such results.*

6. *Is the frontal convolution of serious importance for articulate speech?*

It was certainly not the result of chance that out of the first seventeen cases of aphasia which presented themselves to Broca, in autopsies from the Paris hospitals, sixteen times deep-reaching lesions of the cerebrum were observed, which all had this in common, that they penetrated into the posterior third of the left third frontal convolution, and that in the seventeenth case, together with a destruction of the island and parietal portion, there was present at least a milder anatomical degeneration of the region of Broca. The two first cases of Broca carry with them likewise such an amount of internal evidence as is not often met with in clinical observations.

Case I.—A man, eighty-four years of age, by name Le Long, had an apoplexy, with unconsciousness, one year and a half before his death. Aphasia remained as a sequence until the end—aphasia, without any other paralysis whatever, with good intelligence, good memory and comprehension. He performed properly all voluntary movements of the tongue, lips, etc., and could make himself comprehensible to others by means of gestures and five words, which he uttered somewhat indistinctly, although he was able to articulate distinctly the sounds of which they were composed. We shall return later to the details of the derangement of speech. Broca found a point of softening confined to the posterior third of the left second and third frontal convolutions.

Case II.—In an epileptic, Leborgne, called Tan, because he answered all questions with this little word, aphasia, from the thirtieth to the fortieth year of his life, existed without hemiplegia and with entire power to make himself comprehensible by signs; whether it developed itself slowly or quickly could not be discovered. Then there was added to the aphasia a gradually increasing hemiplegia of the right limbs and a moderate weakness of the right cheek; the tongue moved freely always. In addition to the syllable "tan," Leborgne could, when angry, give vent to a long oath. He died at the age of fifty-one years. The autopsy disclosed softening of the left frontal cortex throughout a wide extent, involving the left island lobe, and the temporal convolution bordering on the fissure of Sylvius; the softening also extended deep into the corpus striatum. Broca made it highly probable that the degeneration arose in the entirely demolished posterior half of the third frontal convolution, confined itself for a long time to this, and then advanced from this point to the other affected portions.

Since then it has indeed been shown that injuries of other cortical regions can also lead to aphasia, without its being necessary that the region of Broca should be involved; but the concurrence of aphasia, with lesions of the left third frontal convolution, has been established with such striking frequency that it cannot be the result of chance. By the side of the observations of Broca just communicated can also be placed many others of no less internal value. One from Th. Simon,[1] *e. g.*, offers almost an experimentum crucis.

A perfectly healthy man fell from his horse. He got up immediately, took hold of the bridle, and was about to vault into the saddle, when a physician, who by chance accompanied him, approached and examined him. He was unable to speak, but made himself understood by signs. No paralysis whatever was present. On the head there was a small wound with depression of the bone. Later, when death ensued from meningitis purulenta and inflammatory softening of the brain, a bit of bone that had been broken off was found in the left third frontal convolution, which, together with the second and the island, was softened. In the skull there was only a perfectly round hole, no crack or fracture in any part.

Another observation from Rosenstein[2] affords the proof that a hemorrhagic softening only as big as a hazel-nut, in the left third frontal convolution, can give rise to aphasia and agraphia as the only symptoms.

A person twenty-two years old, who suffered from diffuse nephritis, suddenly lost in the last weeks of her life the power of speech. The countenance was pale, the look staring, the pupils dilated; she understood what was said to her, put out the tongue when asked, took the drinking-glass when it was offered, tried to answer, but only brought out "ja—ja." She nodded approvingly when one guessed what her wishes were, and tried, when requested, to write; she only scribbled all sorts of scrawls, however, and no letters. She lived sixteen days longer without the aphasia disappearing. Besides the softening already mentioned, there was only found in the brain subarachnoidal œdema. The patient had been for a long time dropsical.

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[1] Berl. klin. Wochenschrift. 1871. Nos. 45, 46, 49, 50. S. 599.

[2] Ibidem, 1868. S. 182.

The table of Lohmeyer, already communicated, gives an approximate representation of the frequency with which lesions of the left third frontal convolution are found in aphasia, in contrast to those of other cortical districts. Out of fifty-three cases of aphasia, there are about thirty-four in which this convolution is affected with other parts of the brain, or alone.

Circumscribed points of softening in other regions of the cortex, with the left third frontal convolution intact, to which aphasic derangements could be referred with the greatest probability, have been but seldom found in the right, more frequently in the left hemisphere. Peter[1] found such a focus in the right hemisphere, also in the posterior third of the third frontal convolution. In the left hemisphere Meynert[2] saw a depot limited to the island and the cortical layer of the operculum. Bernhard,[3] Wernicke[4] and Finckelnburg[5] found the lesions confined to the left temporal lobes; Cornil,[6] Samt,[7] Tripier,[8] and myself,[9] to the convolutions of the parietal lobe. Sander[10] described a case of aphasia in a patient suffering from epilepsy, in which a large spot of softening was seated in the medullary part of the centrum semiovale, in bundles of fibres which he considered to be fibres radiating from the corpus callosum. The mass extended from the central convolution through the parietal lobe as far as the cuneus, and encroached somewhat even upon the medullary part of the frontal convolution. We have already (Chap. XIX., at the end) established, by observations of Farge, Popham, and Jaccoud, the fact that even circumscribed lesions, confined entirely to the white substance of the hemispheres proper, can cause aphasia.

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[1] Bullet. de l'acad. de méd. 1865. 25 Avril. Discours de *M. Trousseau*. The embolical softening reached downward as far as the corpus striatum. Together with aphasia, there was right hemiplegia.

[2] Wien. med. Jahrb. 1866. Bd. 12. § 4. S. 154. *Voisin* also (Soc. méd. hôp. Paris, 1868) saw aphasia with a softening limited to the left island.

[3] Archiv für Psychiatrie. Bd. 4. S. 726.

[4] Der aphatische Symptomencomplex. Breslau, 1874. S. 43.

[5] Loc. cit. S. 3.

[6] Gaz. méd. de Paris. 1864. p. 534.

[7] Archiv für Psychiatrie. Bd. 5. S. 205.

[8] Gaz méd. de Paris, 1874. No. 2.

[9] To this case and to some others of the above-mentioned observations I shall return later with more exactness.

[10] Arch für Psych. Bd. 2. S. 53. Together with aphasia, there was right hemiplegia.

It is only in the case of the occipital lobes that we have not succeeded in finding sufficiently exact observations showing that lesions of this part have led to genuine aphasic disturbances.[1]

The left frontal lobe, and especially its third frontal convolution, possesses, then, by no means a monopoly of producing aphasia, although it is most frequently brought about by lesions at this point. The island comes next in frequency to the frontal lobe. Aphasias, from lesions in other regions, are always simply exceptions to the rule. That aphasias are called into existence by lesions of the occipital lobes, is still to be more satisfactorily proved.

When we reflect how seldom indeed, in general, aphasic derangements proceed from lesions of other regions than the posterior portion of the frontal lobe and the island of the left hemisphere, we shall lay great stress upon this uncommonly frequent concurrence—and the more so when we remember the perfect union which binds all parts of the brain together, and how easily derangements in the circulation, nutrition, and function of one part lead to the same in other parts. When a dyspepsia, a considerable congestion of the head, an annoyance, even, in the case of many persons, impedes the remembrance of words, or causes the stream of thoughts and phrases to stand still, why should we be surprised that such rough onslaughts as hemorrhages, plugging of the arterial courses from embolism, thrombosis, and the like, in different portions of the cerebrum, should be able to throw speech into various sorts of disorders? On the contrary, one should wonder that the gross circumscribed lesions of the brain, which lead to the characteristic aphasic disturbances of speech, are found almost always in one and the same region only. This fact will probably stand out still more distinctly in time, the more thoroughly the cases, in which it is a question of genuine, that is, of ataxic aphasia, are sifted out from those in which there is only a slight word-amnesia, or perhaps a dyslogia.

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[1] The case of *Bateman* (loc. cit. p. 48) which is quoted as an instance of this kind, occurred in a person who suffered from confusion of ideas and weakness of thought; it was a question of dyslogia, and not of aphasia. The quite unsatisfactorily described observations of *Skae* (by *Bateman*, loc. cit. p. 35), and *Magnan* (*Vivent*, De l'aphasie. Thèse, Paris, 1865. Obs. VI.), and others of a similar character, are also quoted.

In consideration of all these circumstances, we venture to make the statement *that the third frontal convolution of the cerebral hemispheres is of vital importance for speech.*[1] But this importance must also probably be granted to the island. We must likewise bring into prominence the fact that *the third frontal convolution and the island of the left hemisphere evidently play a more important rôle in speech than those of the right*, and we must henceforth seek for the grounds on which this surprising difference rests.

7. *Wherein lies the reason of the greater importance of the left over the right hemisphere in speech?*

As we have already stated, Broca[2] connected the strikingly more frequent occurrence of aphasia, after lesions of the left hemisphere, with the use of the right hand by most people, which again is dependent upon their being "left-brained," *i. e.*, exercising the left hemisphere for the more subtile requirements of manual dexterity.

Those whom we style right handed ("droitiers") are left-brained ("gauchers du cerveau"), and vice versâ. The greater skill of the right hand itself Broca deduces from the more rapid development of the left hemisphere, a view which Gratiolet affirms, and C. Vogt and Ecker deny. Moreau's patient, whose case we considered above, could not be right-handed, because from defective development she lacked the left first primitive convolution; she was forced, therefore, to use the right hemisphere in sewing; when she learned to speak, she also must have exercised for this purpose and learned to use the right hemisphere. The same is true of persons whose left hemispheres have been mutilated in childhood, as was the case, *e. g.*, in Parrot's patient, in whom the left island and the third frontal convolution were found destroyed, or in the woman whose brain was sent to us by Dr. Schaefer, of Loerrach.

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[1] *Vulpian* twice found the two left anterior frontal convolutions the seat of softening without aphasia (*Mongie*, De l'aphasie. Thèse. Paris, 1866. Obs. V. and VII.), and their orbital region once (Obs. V).

[2] *Bouillaud* has, moreover, claimed priority of the idea in his report to the Acad. de méd., on the 4th and 11th of April, 1865.

This person had reached the advanced age of seventy years, and died from erysipelas terminating in suppuration of the frontal sinuses. She was an epileptic from childhood, paralyzed upon the right side, and blind of the right eye. She could speak "reasonably well," even a few hours before death; she had "a certain degree of intelligence, a propensity for thieving, and religious comprehension." The right hemisphere is small, all the fissures and convolutions are well marked, although in an almost diagrammatic simplicity. The left hemisphere is for the most part transformed into a loose sac, which is kept in shape by the apparently unchanged pia, and contains a mere shell of nerve-substance from ½ to 1 centimetre thick, half amorphous, half fibrous in its composition. The first frontal convolution had alone survived as a more compact mass, especially in the median and orbital portion; whilst its cranial surface is narrowed, and, at the still recognizable first frontal fissure, loses itself in the above mentioned wall of the sac; in addition, a small supra-orbital, and particularly an orbital, portion of the second frontal convolution is still to a certain extent solid and recognizable; and finally, the gyrus fornicatus can be traced almost as far back as the posterior extremity of the corpus callosum. The parts we have named still show their form by shallow secondary fissures, whilst the surface of the rest of the hemisphere is destitute of any sort of form; and these parts, in common with the central portions of the caudex cerebri and the corpus callosum, which lie exposed at the bottom of the sac and are of normal size, compose a cohesive, more compact mass. The cerebellum seems as little altered in form upon the left as upon the right side.

Whereas for most of the rougher tasks we use both hemispheres, we certainly give preference to the left for most of the more delicate ones. Only in some—for example, in piano and violin playing—do we exercise both; in violin playing, indeed, in very different ways (Wilks).[1] For drawing and writing, we develop the left half of the cerebrum—even the otherwise left-handed people do this; only those with a defective right arm use for these purposes the right brain. Hence, we cannot doubt that *the centres of innervation of the cerebrum are supplied in double for manual tasks, but that most men are, notwithstanding, right-handed, and use the left brain for most manual exercises requiring a certain amount of skill.*

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[1] Guy's Hosp. Reports. Vol. XVII.

Upon this and upon the experiences quoted as to the preponderatingly frequent concurrence of aphasic derangements with lesions of the left hemisphere, rests the hypothesis according to which most men are "*left-brained speakers.*" If this assumption be correct, the following propositions must be justified by experience :—

a. If the portions of the right hemisphere provided for speech, but not functionally developed, are destroyed in right-handed people, speech should still remain intact, since those of the left, which alone have been exercised, remain. If these, however, also fail, then aphasia must be the result.

This state of things has several times been met with. Duval (in Broca), Stewart,[1] and A. Voisin[2] observed persons who, at first the subjects of left-sided hemiplegia, retained their speech, and lost it when right-sided hemiplegia was subsequently added.

b. Left-handed people must become aphasic when the lesions include the right hemisphere. Pye Smith, Hughlings Jackson, and John Ogle have published such cases.[3] Wadham[4] found the same concurrence in the case of a young man who wrote with his right hand, but was in other respects left-handed, as were his four brothers. This "amphidexter" must therefore have used the left hemisphere in writing, the right in speaking, which is indeed surprising, though it strikes us less when we reflect that he learned to speak not only by his own unaided efforts, but also much earlier than he did writing with the right hand, which was compulsory at school.

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[1] Medical Times, July 9, 1864. Circumscribed areas of softening in both fissures of Sylvius.

[2] Gaz. des hôpit. 25 Janvier, 1868. The lesion was situated, on the right side, in the corpus striatum, without implicating the corpus lenticulare; on the left, in the cortex of the island, without participation of the frontal convolutions.

[3] *Simon*, loc. cit., p. 538.

[4] St. George's Hosp. Reports. Vol. IV. p. 245.

William Ogle[1] ascertained the "right- and left-handedness" of about one hundred individuals affected with hemiplegia with implication of speech. Only three were left-handed and paralyzed also on the left side; all the others were paralyzed on the right side.

As organic causes of right-handedness, which he also verified in apes, W. Ogle suggests: greater weight and greater specific gravity of the left hemisphere; greater richness of the frontal regions in convolutions on the left side (in two left-handed women examined by Ogle, the reverse was the case); the earlier fœtal development of the left hemisphere (disputed by Ecker and C. Vogt); finally, the greater afflux of blood to this side. He found the common and internal carotid larger upon the left than upon the right side in twelve out of seventeen right-handed individuals; out of three left-handed persons there was in two no difference, in the third the right common and internal carotid was twice as large as the left.

It follows from this that the repeated observations of destruction of the right third frontal convolution without resulting aphasic disturbances[2] do not invalidate the theory of Broca, since in all these cases there was either right-handedness, or at least the left-handedness was not distinctly established.

Just as little are the generally very rare cases of lesion of the left third frontal convolution without aphasia adapted to refute the hypothesis of Broca, when there was left-handedness or when the right-handedness was not established. But even in those cases where right-handedness is proved, the patient may be in full possession of speech at the time when he came under the observation of the physician, although previously, after the apoplectic attack brought on by the lesion, he was during weeks and months aphasic. Simon[3] had this experience, and I also have seen a similar case. Aphasia, after lesions of the third frontal convolution, is, in fact, in many cases only temporary, and one cannot, therefore, make use of the restoration of speech as a valid argument against Broca, because his hypothesis is protected against this reproach by the "law of substitution." Speech, lost by a lesion of one hemisphere, can return when the individual successfully exercises the other hitherto unused one, as often happens in the loss of facility for writing through paralysis of one hand. The first attempts at writing with the unpractised hand are rude and scarcely legible, just as the first attempts at speech are very imperfect. It is true that the circumstances under which the aphasia is with greater or less facility equalized are not known. One would expect the restoration of speech to take place more rapidly in children and young people than in old ones; still this is by no means always the case.

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[1] Dextral pre-eminence. Philos. Transact. Vol. XLV. p. 279.

[2] See, besides the observation of *Parrot*, quoted on p. 728, the following: *Fernet*, Bull. de la soc. de biol., 1863.—*Mongie*, De l'aphasie. Thèse. Paris, 1866. Obs. 10, 11, 12, 13. All from *Vulpian's* ward.—*Font-Réaulx*, Localisation de la faculté spéciale du langage articulé. Thèse. Paris, 1866. Obs. 19 and 35. Obs. of *Broca* and *Charcot*.—*Simon*, loc. cit., Case 1.

[3] Loc. cit., Case 3.

A very debilitated boy, aged five years and four months, lost his speech, as Duval[1] relates, after a fall from a window, which had as a consequence a depression of the skull. The wound healed in twenty-five days; the boy, however, remained dumb a whole year long, although he seemed very intelligent. There was no hemiplegia. One year after the fall he was drowned. A cyst the size of a nut was found in the left frontal lobe, which was situated, if not entirely, at least in great part, in the third frontal convolution.

We have been able to discover only two observations in the literature, in which lesions of the left third frontal convolution seem to have occasioned no aphasia whatever. They would be of great importance, were it not for the fact that in neither case is it stated whether the patient was right-handed.

Simpson[2] found in an epileptic, who had passed ten years in the Gloucester County Asylum, and in whom neither an apoplectic attack nor a disturbance of speech had ever been observed, an old brownish yellow spot of degeneration in the lower part of the left frontal lobe, which, it is alleged, had destroyed the posterior portion of the third frontal convolution. The cortex was only one line in thickness.

J. Christison[3] relates the case of a woman, who suffered from attacks resembling fainting, after which she spoke constantly in a murmuring way. He found a hemorrhagic spot, the size of a nut, in the left third frontal convolution.

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[1] *Font-Réaulx*, loc. cit., Obs. XXIII.

[2] Med. Times, December 21, 1867. The original unfortunately was not within my reach.

[3] Edinb. Med. Journ. XIX. p. 15. July, 1873.

As things stand at present, we may affirm with Simon that the indispensableness of the third frontal convolution for speech can be regarded as refuted, when—a thing which hitherto has not happened—(1) there should be observed a sudden destruction of the left third frontal convolution in a right-handed person, or conversely of the right in a left-handed person, without the occurrence of an aphasia even for a short time; or when (2) a destruction of both third frontal convolutions should be found in an individual who had spoken up to the time of his death.

Gonzalez Echeverria[1] has published, under the title "Sclerosis of both Anterior Frontal Convolutions without Aphasia," a case which seems to fulfil the second requirement of Simon. But the title is not quite correct, since (1) the person, sixty-seven years of age, an epileptic from his youth, suffered for some months before his death from giddiness upon attempts at walking, accompanied by difficulty in moving the tongue; (2) later he lost entirely at times the power of articulating, while at other times, indeed, he spoke with facility. Derangements of speech, at all events, were not wanting. Then the examination did not reveal a total destruction of the nervous elements composing the frontal convolutions, but only diminution of the same with increase of the connective tissue. We know how even very extensive scleroses are wont to spare a sufficiency of conducting-tracts and nerve-cells.

It is self-evident, however, that while we claim an essential importance in speech for the third frontal convolution, we do not thereby affirm that the cortical functions of speech are only fulfilled through the third frontal convolution on the left side, or exceptionally also on the right. The fact that not only lesions of the third frontal convolution cause aphasic disturbances, but that, in general, lesions of the regions bordering upon the left fissure of Sylvius (the island with the neighboring frontal, parietal, and temporal districts) are those which are almost exclusively involved in serious and permanent aphasic derangements, *indicates the existence in the cortex of a pretty extensive, though still limited, speech-tract. Within this tract, however, we must yield an especial importance to the third frontal convolution.*

8. *Within the cortical speech-tract can centres for the motor*

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[1] New York Med. Record. March 1, 1869.

co-ordination of words and for the acoustic word-images be distinguished from each other ?

One might be tempted to place the motor centre of co-ordination of words in the region of Broca, since it corresponds very nearly to Hitzig's and Ferrier's motor cortical centre for the mouth and tongue, and since the above-quoted cases of aphasia with lesions confined to the frontal convolutions (case Le Long, of Broca, and the case of Rosenstein) can be regarded as of the so-called ataxic character. The separation, which has been attempted by many, of the cortex of the cerebrum into a motor portion situated in front of the fissure of Sylvius and a posterior sensory portion, also suggests the idea of seeking for the workshop of the word-images in the posterior regions. Wernicke succumbed to this temptation. According to his anatomical investigations, the island receives within itself convergent fibres radiating from all quarters of the first primitive convolution, which encloses the fissure of Sylvius. His idea is that it forms the psychical reflex arch between the sensory temporal and the motor frontal portion of the first primitive convolution. The third frontal convolution he regards as the central termination of the nerves of the muscles of speech.

If one tests this hypothesis by means of clinical material, one comes to the conclusion that it finds no sufficient support in absolute experience. An earlier, indeed, and therefore not entirely reliable, observation of Lallemand,[1] seems to argue that even pure ataxic aphasia can proceed from the "middle lobe," whilst a very exact one of Sanders[2] at least proves that softening confined to the posterior part of the left third frontal convolution and the under part of the anterior central convolution may, indeed, at first give rise to a complete loss of speech, leaving behind later only an advanced degree of word-amnesia. As for the island, an observation of Meynert's,[3] which leaves nothing to be desired on the score of anatomical accuracy, but much clinically, shows that an acute encephalitic softening of the extreme posterior portion of the left island and of that portion of the cortical layer of the operculum which approaches the surface of the island, is sufficient to produce word-amnesia.

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[1] *Falret*, Arch. gén. de méd. Vol. LXXXIII. 1864. T. 1. p. 344. I shall return to this case in the next chapter.

[2] Edinb. Med. Journ. 1866. Vol. XI. T. 2. p. 811. A woman 43 years old suffered from heart disease, and the softening was presumably of the nature of embolism. She succumbed to gangrene of the leg.

[3] Wiener med. Jahrb. 1866. Bd. XII. § 4. S. 154.

A girl, twenty-two years old, who suffered from insufficiency and stenosis of the valves of the left heart and aorta, was suddenly seized with aphasia two weeks before her death. With complete mobility of the tongue, she was unable to find certain words, or used wrong ones, *e. g.*, "yellow" instead of "hands." "Husten" she changed into "hutzen."

A prudent abstention from an answer to the above proposition is, therefore, at present to be recommended. It is, notwithstanding, remarkable that (1) *the anterior regions of the cortex, either alone or in connection with the posterior, are almost without exception found affected in cases of pure ataxic or mixed amnesic and ataxic aphasia; and that* (2) *in pure amnesic aphasia sometimes the anterior, sometimes the posterior cortical regions alone appear affected.* In the following chapters we will still further establish these facts by numerous proofs. The latter statement accords with the known experience that of all the functions of speech the remembrance of words is the most easily disturbed, and by the most varied lesions of the brain.

Piorry[1] reports the case of an old priest, who, after a stroke of apoplexy, could no longer find nouns, and the autopsy revealed three old apoplectic cyst in the anterior portion of the left corpus striatum. "Voulait-il demander son chapeau, il se servait de verbes, de pronoms, d'adjectifs, pour rendre son idée: Donnez-moi mon . . . ., ce qui se met sur la . . . ., mais le mot tête ne lui venait pas," etc. If the cortex was, indeed, uninjured in this case—one must not forget that Piorry quotes an old case from memory—we must not conclude from it that the power of remembering words is to be sought in the corpus striatum; but the case admits of the interpretation that the lesion of the corpus striatum reacted in a disturbing manner upon the functions of those portions of the cortex in which the recollection of words is accomplished.

When we call to mind the completeness of the nutritive union which exists between different portions of the brain (see p. 737), when especially we again consider that the function of one portion of the cortex can without doubt be impeded by the serious irritation of another portion functionally connected with it, the fact that the genuine ataxic aphasias proceed almost exclusively from lesions of the anterior cortical regions, and especially of the third frontal convolution, makes it in a high degree probable that the motor co-ordination of words takes place here. Clinical experiences up to the present time enable us to draw no conclusion as to the regions in which the acoustic word-images are evolved and enter into connection with the corresponding conceptions.

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[1] Bull. de l'acad. de méd. 1864-1865. T. XXX. p. 793.

An extremely valuable observation of Cornil,[1] with very exact anatomical details, seems to us capable of being thus interpreted, viz., that the inflammatory irritation of a posterior portion of the cortex had reacted with a disturbing and interrupting effect upon the motor co-ordination of the anterior regions; thus lending support to the proposition above laid down, that the irritation of one portion of the cortex can interrupt the activity of another portion functionally connected with it. The disturbance of speech observed in this case we might designate as *aphasic stuttering*.

A young man with phthisis fell a victim to that disease nine weeks after a light stroke of apoplexy, which had left behind a great derangement of speech lasting several days, together with an anæsthesia of the skin likewise lasting only a few days. The patient made every effort to utter words; he remained, however, incomprehensible, stuttered, broke off, and began his attempts to speak anew without success. The muscular sense had not suffered upon the anæsthetic side. An inflammatory focus, measuring twelve centimetres square, was found in the left second posterior parietal convolution.

Finally, we will point still again to the important fact, which increases the exceeding difficulties of localization of the functions of speech, that sometimes—although not frequently, it is true—we are unable at the autopsy to discover any gross lesion of the brain after a loss of speech occurring suddenly amidst severe cerebral symptoms. Gairdner,[2] of Glasgow, communicated such an observation.

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[1] Gaz. méd. 1864. p. 534.

[2] Glasgow Med. Journ. May, 1866.

A man was visited by an epileptic attack, after which loss of speech and a sort of cataleptic condition remained behind, without coma or evident paralysis. The intelligence returned in great measure, but the speechlessness continued. The patient was incapable of expressing his thoughts in words, but copied written documents, with tolerable accuracy. After ten weeks he died during an epileptic attack. The autopsy disclosed nothing except a general and diffuse congestion of the pia mater.

CHAPTER XXIV.

The Two Great Classes of Cortical Derangements of Speech: Dysphasic and Dyslogic.—The Conceptions of Aphasia in a Clinical or Practical Sense, and of Aphasia in a Theoretical Sense in Reference to Speech, do not cover each other. —The Aphasia of Practitioners includes also Verbal Anarthria.—Review of the Cortical Dysphasic Derangements, which Clinical Teachers include within the Frame of the Sketch of Aphasic Disease, and of those which they exclude.

The conception of the cortical derangements of speech is broader than that of the dysphasic. The cortex is not only the organ of speech, as it is uttered in words grammatically formed and arrayed in sentences according to laws proper to them, and independent of the individual Ego, but it is also the organ of the intelligence, which stamps upon speech its personal character. When the operations of the mind suffer disturbances, then these find their corresponding expression in the speech, and these secondary derangements of speech proceeding from the diseased "Intellectorium" we style dyslogic, or *dysphrasias*. Hence, the cortical derangements of speech may be separated into two great classes: *dysphasic* and *dyslogic*, or *dysphasias* and *dysphrasias*. We will first examine more closely the dysphasias, and later cast a glance over the dysphrasias.

We have previously (Chap. IX.) divided all derangements of speech as an independent faculty, into those of articulation and those of diction; the former we have designated as *dysarthrias*, the latter as *dysphasias*. If we hold fast to this, then we must allow also *cortical dysarthrias*, for the cortex forms words not only as acoustic symbols for conceptions, but also as motor aggregates of sound; it imparts centrifugal impulses, by means of which the word is handed over as an organized motor unit to the infracortical organs of articulation for realization. Clinical medicine, however, has paid no regard to this point. It briefly styles every cortical inability to form words *aphasia*, regardless whether diction or articulation is interfered with, and contrasts with each other ataxic and amnesic aphasia. The former it regards, after the example of Popham and W. Ogle, as an incapacity for the motor co-ordination of words; the latter as an inability to call to mind words as sensory forms or acoustic sound-pictures. In amnesic aphasia there is lacking the internal word, that which is comprehensible to the speaker alone; in the ataxic form, the production of the external word is affected—the word which is comprehensible to others, as it is given forth from the co-ordination of sounds as motor forms through the cortical key-board of the will. Obviously Steinthal [1] is right, when he disputes the dysphasic character of the so-called ataxic aphasia, so long as mere derangements of simple diction are allowed to pass as aphasia.

Strictly taken, ataxic aphasia is nothing else than cortical anarthria of words, *verbal anarthria;* amnesic aphasia, on the other hand, is true aphasia, *incapacity of verbal diction.* When, however, we hereafter designate the cortical anarthria of words as ataxic aphasia, this inconsistency is justified partly by the fact that this last designation has grown too firmly into medical forms of speech for one to hope to be successful in wresting from it its citizenship, partly by an important practical consideration. The cortical inability to form words is in very many cases of a mixed nature; not only the cortical articulation of the word as a motor sign suffers, but also its recollection as an acoustic sign, and often nothing is more difficult than to distinguish in a given case where the dividing-line is drawn between diction and articulation.

Further, we have already (Chap. VIII.) called attention to the fact that under aphasia, in the clinical sense, a great quantity of symptoms are embraced, which are of a very varied nature. A single picture of the disease has been constructed, whose features were gradually put together from numerous separate observations not agreeing at all with each other, and without a thorough comprehension of the elementary processes, which constitute speech.

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[1] Abriss der Sprachwissenschaft. S. 454.

In the main it is the following dysphasic derangements which are comprehended under the general name of *aphasia:*

1. *Ataxic aphasia*, or incapacity for the motor co-ordination of words.

2. *Amnesic aphasia*, or incapacity for the recollection of words as acoustic aggregates of sound.

3. *Dumbness for words*, or the inability, with good hearing and sufficiently preserved intelligence, to understand words as well as previously.

4. *Paraphasia*, or the inability to properly connect word-images and the corresponding conceptions, so that, instead of the ones corresponding to the sense, misplaced or entirely incomprehensible word-images present themselves.

5. *Agrammatism* and *akataphasia*, or the inability to form words grammatically and to arrange them in sentences syntactically.

These embrace all the aphasic derangements recognized by clinical teachers, but not all the actually occurring cortical derangements of the autonomous function of speech.

Thus there exists without doubt *a retarded and a hastened form of speech*—the latter, up to the point of indistinctness from interference of the words, both forms having their origin in an abnormal course (as to time) of the cortical excitation which forms the basis of the elementary processes, out of which speaking is developed by diction and articulation. We frequently observe in meningitis and in different diffuse cortical affections a strikingly measured speech, which we may designate as *bradyphasia;* at other times, in conditions of excessive cerebral excitation, a "*tumultus sermonis*," increasing even to incomprehensibility, which admits of the interpretation already given.

A recent observation makes it seem to us even probable that *scanning utterance* has at times a cortical origin, for we saw it in the case of a young man during convalescence, who, in consequence of a depression of the left parietal bone, remained for weeks speechless after the intelligence with comprehension of words had been rapidly re-established, and after he had written down with the unparalyzed right hand incomprehensible lett and words. At this time, as a convalescent, he spoke all wo properly, but slowly and scanningly; he also wrote them do properly, but slowly. He moved the tongue in all directio but at the same time somewhat slowly.

We have, moreover, previously made mention (p. 746) of *aphasic stuttering*, which we have good ground to regard a cortical derangement.

Here also belongs that *stumbling over syllables* of wh we have already repeatedly made mention (Chaps. XIII. a XVII.). We will return to it in an especial chapter.

In the first place, then, let us carefully consider the d phasias which the practitioners embrace under the general na of aphasia. We shall join to this the consideration of the *d graphias* or derangements in written speech analogous to dysphasias, and which one generally finds described under names *agraphia* or *paragraphia*. The disturbances in speech of gestures (Geberdensprache), or *dysmimias*, will a be taken into consideration.

Before we begin this task, however, with a descriptio ataxic and amnesic aphasia, we must again premise the rem that we regard both of these designations, against which we h already raised objections and which we retain only for objec reasons, as not happily selected. Ataxic aphasia is in a cer sense also amnesic, and amnesic aphasia ataxic.

If, for example, one considers memory as a general func of the nervous system, then there must be a recollection of acoustic as well as of the motor word-images, by means of wh the co-ordination of sounds into words is accomplished. recollection of words is therefore a double affair: 1. *a recollec of words, in so far as they are acoustic combinations of soun* 2. *a recollection of words as movement-images*. Trousseau always referred aphasia to a loss of recollection at times of word-signs, at times of the action by means of which words are articulated, and always appreciated this correctly. W. O also distinguished two kinds of recollections of words: in a

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St. George's Hosp. Rep. V. 2. 1867, p. [illegible] tion to that which one ordinarily understands by this term, and by the aid of which we are conscious of a word, he recognized a second also, by means of which the word is pronounced. What is called ataxic aphasia is therefore always of an amnesic character.

On the other hand, amnesic aphasia leads always likewise to ataxia, since the co-ordination of words is determined only in part by the movement-images; the acoustic word is also necessary thereto — it regulates the movements, but from a more distant station. If the word as an acoustic sound-structure, or the connection between the acoustic and motor centres, experiences some disturbance, then disordered, confused, or mutilated words must come to the front. Consequently, we may with Bastian[1] even regard paraphrasia, which has its basis in a disordered connection between the centre that gives birth to ideas (Maudsley's Intellectorium) and the acoustic word-centre, as an ataxic derangement of diction, or a dysphasia.

CHAPTER XXV.

Ataxic Aphasia, Agraphia, and Amimia.

There are cases of *entire loss of speech*, in which the patients retain the words in the memory as acoustic signs, and still are absolutely incapable of uttering them, although the intellect may be clear, and the movements of the tongue free. That they really are in full possession of the acoustic word-signs is clearly evident from the fact that they can translate the same into writing. If, however, they be requested to repeat the sounds or words from dictation, it is impossible for them to comply, even when they are shown how the tongue and lips should move. They open the mouth, twist the lips, and make grimaces, but bring forth, at the best, only inarticulate sounds and grunts.

Thus, Trousseau[2] tells of a robust young civil functionary, who, in an attack of unconsciousness, lost the power of speech entirely, although there was no paralysis of any parts of the body. He could execute all the movements of the tongue and lips with the greatest facility. He was able to discharge the duties of his office, notwithstanding the loss of speech, as he could transact all his business in writing, and he delivered to Trousseau an exhaustive history of his illness, that had been written out by himself.

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[1] Brit. and Foreign Med.-Chir. Rev. 1869. pp. 209 and 470.

[2] Clinique Médicale. Tome II. Art. Aphasie.

Bouillaud[1] was present, in the year 1828, at the autopsy of a young man who died in the surgical ward of Phil. Boyer. An umbrella had been struck with such force into the left orbit, that the eyeball protruded. During the eight days that he survived, the power of speech was entirely lost, although he could still move his tongue. He understood what was said to him, but was unable to answer. On the other hand, he wrote down his wishes on paper, and stated in writing that he retained his memory, but could not utter the words. There was found a suppurative softening of the left frontal lobe, which had been bored through by the point of the umbrella, the perforation extending as far back as the anterior end of the left lateral ventricle. The end of the umbrella must, according to the direction of the wound, have penetrated the third frontal convolution.

Boinet[2] relates the case of a man in whose brain a traumatic abscess the size of a nut was found in the region of Broca's convolution, and extending as far as the corpus striatum. There was no secondary softening of the surrounding parts, but there were traces of a very circumscribed meningitis. After the healing of a wound made by the trepan, the patient had regained intelligence and memory, but remained speechless. He made himself understood by gestures, played with his comrades, copied from dictation, and wrote out his own thoughts. In addition to the speechlessness, there was slight facial paralysis.

At other times the patient is *not entirely speechless;* he can still pronounce some *words of one syllable*, but has to resort to writing when he wishes to use any other words.

Thus, in the case reported by Lallemand, which is mentioned on page 744, the man, who was fifty years of age, retained his intelligence and the power of executing all the movements of the body, those of the tongue among the rest, but his speech was reduced to a few monosyllabic words. He could, however, make himself understood, as far as his education permitted, by writing. Lallemand found a circumscribed spot of softening on the surface of the middle lobe.

Written speech constitutes the most valuable and the most reliable, though not the only means of proving that the memory for spoken words is retained. We must assume the same in those cases in which aphasic persons, who have never learned or have forgotten the art of writing, are able to give expression to their thoughts and to answer questions correctly by expressive gestures. It is true that it is easy to be deceived in such cases, and the observer must convince himself by careful and repeated examinations, that the patients really understand him. He must himself especially avoid the use of any explanative gestures in connection with his words. We possess observations which place the existence of an ataxic aphasia under such circumstances beyond doubt. Out of them we may create a *second variety* of this form of derangement of speech.

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[1] Bull. de l'acad. méd. T. XXX. p. 625.

[2] Gaz. des hôpit. 1872. No. 30. p. 235.

Many of these patients possess still a small stock *of words*, which they turn to good use in making their gestures more intelligible. Others utter only *a few senseless, and often very extraordinary syllables and words*. The *repetition* of words from dictation is impossible in ataxic aphasia, even when the patient still retains control of a small stock of words. In this the ataxic differs from the amnesic form of aphasia (Ogle). We do not, however, mean to say that all persons with amnesic aphasia can repeat dictated words; the majority of them can, but a very severe derangement of the recollection of words abolishes likewise the ability to repeat words from dictation. Further, patients with ataxic aphasia are unable to form other combinations with the syllables and sounds of the words they are still able to enunciate (Trousseau). One aphasic person, who could say "Bon jour, monsieur" very well, could not pronounce the word "bonbon" at all (Perroud). Another, whose vocabulary consisted almost solely of the odd word "cousisi," could not utter the word "coucon" or "sisi" (Trousseau). On the other hand, they are sometimes able to give vent to even long and well articulated oaths; we have already (Chapter XVI.) endeavored to give an explanation of this strange fact. The patient who answered almost all questions with the word "cousisi" also employed the mutilated word "saccon," which was evidently meant for "Sacré nom de Dieu" (Trousseau).

The case of Leborgne, reported by Broca (Chapter XXII., p. 734) illustrates the last-mentioned fact. Leborgne accompanied his demonstrative gestures with the diminutive "tan," or its reduplication "tan, tan;" when he was not understood, he became enraged and gave vent to a long and savage oath.

The case of Le Long (Chap. XXII., p. 734), also reported by Broca, is an excellent example of a *defective vocabulary with constant and unvarying mutilation of some of the still remaining words*. This case shows how, in ataxic aphasia, the same sound may be constantly employed in one word, and just as constantly left out in another, a proof that it is not the articulation of the sound in itself which has become an impossibility, but its combination with other sounds in the word. It is the construction of the word and not the production of the sound, that is prevented. Both defects can, it is true, occur together, producing mixed anarthric-aphasic derangements.

Le Long had at his disposition only five words, with which he supplemented his very expressive gestures: oui, non, tois instead of trois, toujours, and Le Lo instead of Le Long—that is, three perfect and two mutilated words. He affirmed with oui, and denied with non; for all numbers he employed tois, indicating the exact number which he had in his mind by an adroit use of his fingers; he designated himself by Le Lo; he used the word toujours when he could not indicate his meaning by any of the other words.

Thus Le Long always pronounced the r in toujours correctly and elided it in trois, as children do who have not yet mastered the difficulty of the conjunction of the r with the preceding t; he had permanently lost the power of articulating that combination of sounds. The nasal sound, which he articulated in non, he could no longer add at the end of his own name.

When *agraphia* accompanies ataxic aphasia, it is sometimes *absolute* and *literal*, the patients being unable to produce a single letter with the pen. They scribble lines to no purpose upon the paper, and finally admit unwillingly their inability to write. Others can still write letters and entire rows of letters, separated here and there in the semblance of words by little spaces; these combinations of letters, however, are for the most part unpronounceable: *verbal* agraphia. It is true that among the unpronounceable rows of letters, we also come across written words that can be articulated and that have meanings; but it is, as a rule, impossible to discover from them what the patient wishes to say. The words that are most frequently written correctly are their own names, but even these are often mutilated. When they are able to perceive that they have forgotten how to write, and that the signs on the paper do not express their thoughts, the affection is ataxic agraphia. If, however, they continue to write, without being conscious of these facts, we have no longer to deal with a simple ataxic agraphia; in such cases the connection between the idea and its written-image is broken, or the written-images are obliterated in the memory. As the aphasic patient can use his tongue for all purposes except for speaking, so the agraphic patient can still use his hands in all sorts of delicate occupations, with the sole exception of writing. Spamer, *e. g.*, saw in Giessen a young agraphic woman who could sew very skilfully.

It is evident from these facts, *that the centres of co-ordination for spoken and written words are different and separated from each other in position* (Marcé).[1] As we proceed we will detail many more observations which prove that derangements in written and spoken speech do not always run parallel. Since, however, both faculties are, as a rule, deranged together, it follows *that the two centres are at all events closely united, and that their tracks interlace.*

The *sign-speech*, or the *speech of gestures*, is more rarely affected. The aphasias, in which the power of expressing the thoughts by means of gestures is impaired, are always of an intense and complicated nature. Here, too, we meet with patients who are aware of the fact that the power of pantomimic expression is lost, while others remain ignorant of the loss.

A woman, sixty-one years of age, was struck with apoplexy on January 3, 1862. After the coma disappeared, she suffered from hemiplegia and hemianæsthesia of the right side with aphasia. She could pronounce occasionally and with effort the word mami, which she used in answer to all questions, and now and then the word non, which caused her still more trouble. Gesticulatory speech was impaired. She nodded affirmatively with the head when she wished to answer in the negative, or raised up two fingers in order to express four, or five instead of three. It was evident from the expression of her face that she knew very well that she expressed herself incorrectly. She died Dec. 15th.

An apoplectic cyst embraced a great part of the third left frontal convolution, the entire left island, and the neighboring medullary substance along with the anterior third of the corpus striatum (Perroud).[2]

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[1] De l'existence d'un principe coordinateur de l'écriture. Mém. de la soc. de biol. II. série. T. III. 1865.

[2] Journ. de méd. de Lyon. 1864.

A woman was paralyzed on the left half of the body, and aphasic from apoplexy. She still had at her disposition only the following little phrases which she uttered with interjectional sprightliness: "Oui, parbleu!" "Tiens!" and "Vous comprenez?" When asked whether she wished to eat, she answered: "Oui, parbleu!" what she wished to eat? "Oui, parbleu!" what her name was? "Oui, parbleu!" or also "Tiens!" in a mocking and snappish tone. She seemed persuaded that her answers were to the point. She often added, "Vous comprenez?" in the tone in which a person would use it who thinks he has convinced the person speaking with him. She often made use of gestures, which were as limited and senseless as her discourse (Peter). Here there was amnesic or combined amnesic and ataxic derangements.

Trousseau found also that aphasics sometimes imitate gestures, but cannot make them at request, unless they have been previously made before them. This is not ataxic, but *amnesic amimia.*

The patient Paquet, who, besides his name, could pronounce almost only the word "cousisi," imitated the movements of clarionet-playing with exactitude, and comprehended also what they meant. When requested a few moments later to make the same motions of clarionet-playing, he reflected, but was generally incapable of accomplishing this simple pantomine.

We will close this chapter with an abstract of a case observed by Westphal, in which there was in our opinion aphasia of a decided ataxic nature. It is impossible to determine from the very meagre account of the case that has been published whether it was accompanied by amnesia. What seems to us especially interesting in this case is the inability to utter *words* with the exception of two senseless word-like combinations of sounds, while the patient, under certain circumstances, was still able to articulate *letters.*

A man in Westphal's[1] clinic, who shortly before had been paralyzed by an apoplectic stroke, had the appearance of a person of great intelligence, but was unable either to utter a word *spantaneously*, or to *repeat one after another person.* When a word was dictated to him, he opened his mouth, made all sorts of grimaces, and visibly exerted himself to respond to the demand, but always gave utterance instead to the sounds "tschi-tschi," or "akoko." He was also unable to repeat certain sounds. When he was told to read aloud from a book—he had learned to read

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[1] Zeitschr. f. Ethnologie. Bd. VI. 1874. Verhandl. der Berliner Gesellsch. f. Anthropologie. S. 94.

—he brought forth in a spelling manner (no matter what the letters were in the text before him), the sounds a, u, æ, etc., which he was able, on request, to repeat afterwards. When told to put out his tongue, he opened his mouth, without putting it out at first. At other times, however, he succeeded in protruding the tongue, which then showed itself to be freely movable. Upon dictation he wrote his own name and that of his wife correctly.

CHAPTER XXVI.

Amnesic Aphasia.—Simple Aphasia of Recollection (Erinnerungs-Aphasie), and Aphasia with more intense Disturbances of Memory.—Influence of Word-Amnesia and of Aphasia in General upon Mental Activity.—Complicated Forms of Amnesic Aphasia.

As Biermer[1] has very appositely remarked, the converse of the Mephistophelian verse: "When the ideas fail, a word comes opportunely to hand,"[2] applies to *amnesic aphasia. The idea is present, but the word is wanting, although articulation is at the service of the word.* The idea of an object, or of its properties and relations, rises up in the mind, but the accompanying word-image is wanting, or only partially enters the recollection.

Two things are possible here. Either the word is entirely extinguished in the memory, or it still remains there, but does not get afloat; or, to use a more common expression, *the association between word and idea is interrupted.*

Altogether the most frequent form of aphasia is that in which the word is still preserved in the memory, but the idea can no longer bring it into recollection, while it immediately emerges and can be correctly spoken, as soon as it is either spoken or read before the patient.

Nasse[3] and after him Falret[4] have made from this a special class of aphasics, viz., those who, the intellect being intact, cannot recall certain words, classes of words, and letters, but can still repeat and also write out all words which are spoken before them. This is *simple aphasia of recollection.*

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[1] Vortrag über Aphasie in Zürich, 1 Jan. 1871. Correspondenzblatt für schweiz. Aerzte I. 8.

[2] "Wo die Begriffe fehlen, da stellt ein Wort zu rechter Zeit sich ein."

[3] Allg. Zeitschr. f. Psychiatrie. Bd. 10. 1853. S. 525.

[4] Arch. gén. de méd. 1864. Vol. I. p. 339.

Thus, an epileptic could not utter a single word spontaneously, but he repeated and wrote without difficulty words that were spoken before him. (Forbes Winslow.[1])

Proper names and substantives in general are the words which are most frequently forgotten; sometimes verbs, adjectives and pronouns are also forgotten, and occasionally all words have slipped from the memory. It is usual to illustrate the forgetting of proper names by Crichton's well-known anecdote of the ambassador at St. Petersburg, who, when asked his name on the occasion of making a visit, had first to call out to his attendant, "For heaven's sake, tell me what my name is!"

When the aphasic patients do not find the substantives they want, they paraphrase them in such a way that the integrity of the idea can be clearly recognized.

A hind, forty years of age, was unconscious for four weeks after a severe injury of the head; he regained his recollection of things and places, but his memory for names was lost. The nouns had disappeared from his vocabulary, but he still had command of the verbs. A pair of scissors he called that with which one cuts; the window, that through which one sees, through which the room is illumined, etc. He had forgotten most of his songs and prayers. He recovered his memory subsequently (Bergmann [2]).

A case observed by Graves,[3] a thoroughly trustworthy authority, is uncommonly interesting, since it shows that only the *initial letters* of the substantive, and consequently *only a portion of the word*, may be aroused in the memory by the idea, the rest of the word not emerging until the corresponding written word meets the eye.

A man fifty-six years of age, after an apoplectic attack, lost his memory for proper names and substantives in general, with the exception of their first letters, although the power of speech was not impaired in other respects. He prepared for himself an alphabetically arranged dictionary of the substantives required in his home intercourse, and whenever it became necessary for him to use a noun, he immediately looked it out in his dictionary. When he wished, *e. g.*, to say Kuh (cow), he looked under K. As long as he kept his eye upon the written name, he could pronounce it, but a moment afterwards he was unable to do so.

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[1] Obscure Diseases of the Brain and Mind. p. 510.

[2] Allg. Zeitschr. f. Psychiatrie. 1869. Bd. 6. S. 547.

[3] Dublin Quarterly Journ. T. XI. p. 1. 1851.

On the other hand, a boy, whom L. Schlesinger[1] observed, left out the initial consonants of words in speaking and writing.

The boy, who was eight years of age, became speechless in consequence of a commotio cerebri, after which he had been unconscious for six days. Immediately after the return of consciousness he could make himself perfectly intelligible by signs, but could neither speak nor write. He gradually learned both again. One month after the receipt of the injury he was able to speak nearly everything, but left out invariably the initial consonants of the words. He spoke and wrote, *e. g.*, "Ich ar icht ort," instead of "Ich war nicht dort."

The question now arises, why *nouns*, and especially *proper names* and *names of things*, are more readily forgotten than verbs, adjectives, conjunctions, and the other parts of speech? The more concrete the idea, the more readily is the word to designate it forgotten, when the memory fails. Probably the only reason for this is that the conceptions of persons and things are more loosely connected with their names than the abstractions of their circumstances, relations, and properties are. We easily represent persons and things to ourselves without names; the image of sense is here more essential than the symbol, *i. e.*, the name, which conduces but little to our comprehension of personages or objects. *More abstract* conceptions, on the contrary, are attained only with the aid of the words, which alone give them their exact shape. Hence verbs, adjectives, pronouns, and, still more, adverbs, prepositions, and conjunctions, possess a much more intimate relation to thought than nouns. We can conceive that the processes of excitation and the combinations in the cellular networks of the cerebral cortex must be much more numerous for the creation of an abstract than of a concrete conception, and that the organic tracts which connect the former with its name must be correspondingly much more numerous than those of the concrete.

This accounts for the fact that forgetfulness of the names of persons and of things is, even in the fulness of health, a very common occurrence. Gradual transitions lead from this *physiological* to *pathological* word-amnesia. Elderly people frequently complain of the failure of their recollection for names at a time, perhaps, when they think with the greatest vigor, and long before the *morbid senile amnesia* sets in. In riper years, persons and objects interest us less on their own account than on account of the associations bound up with them, the concrete phenomena less than the ruling idea and the principle governing the phenomena. The capacity to grasp and to hold fast in the memory the laws by which facts are regulated, increases as we advance in years, while the memory for words diminishes. In *senile amnesia*, provided it is really *morbid*, the former suffers also. Past events, even important ones, vanish from the memory; things that occurred during early life remaining fresh in the recollection, when more recent occurrences have been entirely forgotten. The patients forget the names of their best friends and even of their own children; at last they can only recognize their nearest connections with effort, and gradually fall into a condition of *senile imbecility*.

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[1] Wiener med. Presse. 1869. Nr. 37.

Let us now contrast *the more profound derangements of memory*, which, as a rule, are of a complicated nature, with the aphasia of recollection. In the former we do not have to deal with a simple loosening of the bonds between the idea and the word. On the contrary, the words are shattered in their structure as acoustic combinations of sound, and are sometimes obliterated completely. In this condition also it is the proper names and substantives which suffer in the first instance, and the verbs, adjectives, and pronouns in the second, while the parts of speech which represent rather the framework of language are still unaffected. The patient distinguishes and understands the words when they are pronounced before him, and endeavors to repeat them, but with very varying, and, as a rule, only partial success. When the patient is anxious to learn, and pays strict attention, fastening his eye upon the mouth of the speaker, and the word is repeated before him several times, he can often reproduce it correctly. Otherwise it cannot be uttered at all, or is articulated in a mutilated shape; sometimes this, sometimes that sound or syllable is forgotten, or the sounds and syllables are incorrectly combined, or alien syllables which belong to kindred words are interpolated or added in place of the final syllables. The mutilation and distortion of words occurs in varying forms, now in this form, now in that, and not in the constant form we find in ataxic aphasia. At the same time the articulation of the sounds is unaffected, provided the case be not complicated by literal anarthria. The written language is invariably affected, and, as a rule, to a much greater degree than the spoken language. The letter-signs may be entirely forgotten, or can no longer be arranged in the form of words. In some cases a few written word-images still emerge from the depths of the memory in a recognizable and correct, or in a mutilated and contorted shape.

The following case, which came under my own observation, and along with which I give the results of the autopsy, will illustrate for the reader this form of severe amnesic aphasia.

A stone-mason, fifty-five years of age, with extensive atheromatous degeneration of the arteries, remained a long time in the surgical clinic in Freiburg, where he was treated for ulcers of the feet. On March 19th, 1875, these had cicatrized. Up to that time no disturbances of the intelligence or of speech had been remarked. On the evening of March 22d, a weakness of the right arm, and a transient inability to find words, suddenly set in; for some days previously he had suffered from loss of appetite and nausea. At noon on March 23d the aphasia returned and this time was permanent. In the evening there was paralysis of the right leg. The patient was transferred to the medical wards. On the following day we found paralysis and anæsthesia of both legs, with cessation of the circulation in both crural arteries. These serious symptoms were caused by thrombosis of the abdominal aorta, and resulted in death on April 12th, from mummification of the legs. We had therefore an interval of three weeks in which to study the patient's aphasia. The paralysis of the right arm disappeared sufficiently in a few days (on March 26th) to allow the patient to use the arm for the purposes of eating, and it gradually disappeared almost entirely. No anæsthesia of the arm could be discovered. In the face and tongue there was no sign of paralysis.

The patient had lost to a great extent the ability to find words, but not the power of articulation. He repeated all the letters correctly, with the exception of the letter-word ypsilon (y), of which he could only repeat the syllable yps. He could not repeat the alphabet spontaneously; sometimes he began to count instead of to spell, and broke off in a pout when he discovered his error, and at other times he would begin in a careful and resolute manner, and succeed perhaps in bringing forth several, even six or eight letters consecutively, but would then become silent or utter a medley of letters, some of which had been already spoken and some had not. He was still able to reproduce in imitation simple syllables and even words of two or three syllables, but would say, for instance, "bobe" instead of "bebo," etc.; words of more than three syllables he could not utter. Instead of "Constantinople" he said "Stozati, Stozate, Stozatalsch." After prolonged schooling, in which he was made to pronounce one syllable after another, and to watch closely at the same time the mouth of the person speaking to him, he achieved "Constanti," but could get no further. In counting he proceeded sometimes as far as twelve, sometimes as far as sixteen; then followed complete confusion, in which he at times perceived that he was astray, at times not.

His name, Senn, he found and pronounced on the second attempt; the name of his birthplace, Buchheim, only after several fruitless efforts and with assistance from others. To the question in which county the village lay came the answer "Here" (Buchheim is situated in the county of Freiburg). When asked the name of the county town which he meant by "here," he answered in an animated tone, "I shall find it yet." He was unable to find the name, however. When the word "Freiburg" was pronounced before him, he said "feig-burg-burg-frei-fro." He was made to fix his attention on the mouth of the speaker, and at last he succeeded in saying "Freiburg." He was then asked in what country Freiburg lies? He answered, "Of course I know that!" but he was unable to find the word for it. "Grossherzogthum Baden" was pronounced before him, and he said, in attempted imitation, "grossherzog, grossfrei." He could not say knife, fork, etc., although he knew them. We tried to teach him the names for them, sometimes with success sometimes without. Instead of "Gabel" he said "Gasser," instead of "Löffel," "Flöfe." He wrote his name with his shaky right hand, but wrote "Sen" instead of "Senn," forgetting therefore the second "n;" when the mistake was pointed out to him, he was unable to correct it. Of his first name "Friedrich" he could only say "Fri." He was unable to write down his age and birthday. We could not, on account of the marked presbyopia of the patient, ascertain to what extent he was able to understand what he read.

When the patient attempted to speak of his own accord, he was unable to find the words. He contented himself therefore with indicating his meaning by gestures. He still possessed, however, a small store of words and sentences, of which he made constant use. They were the interjections: "O, je!" "O, weh!" "Mein Gott!" "Maria Josef!" "Das ist zu arg!" (that is too hard), "Ja freilich!" and now and then also: "Sacrament!" He summoned persons by "Sie!" (you). He answered questions always correctly with "Ja!" (yes), or "Nein!" (no), or "Zu viel!" (too much), "Zu arg!" (too bad), "Nicht so arg!" (not so bad). He took the liveliest interest in everything that took place in the ward.

On the 6th of April the patient began to be delirious when left alone. At this time he had fever. He uttered undistinguishable syllables which were frequently reiterated, along with the same expressions of wonderment and words of complaint as before. After April 8th he threw in occasionally French words. Between the "Maria Josef," "Das ist halt so!" "Das ist's eben!" "Es ist halt zu arg!" he would suddenly strike in with "Mon Dieu! mon Dieu!" When requested, *e. g.*, to designate the seat of the pains he complained of, he answered, "Pas toujours!" Immediately afterwards he would reply to another question in an irritable manner, but still in words that showed a correct understanding of the query: "O, je n'en suis pas sûr." We ascertained, on addressing him in French, that he had lived long in France. He always answered correctly with: "Oui, monsieur!" "Oui!" "Non!" "Mais si!" "Pas trop!" "Bien!" and thanked with "Merci!" To the question: "Vous étiez à Paris?" the answer was, "Si, j'y étais." Question: "Was er dort getrieben?" (what he did there). Answer: "J'ai pait .... !" unintelligible syllable. Question: "Avez-vous travaillé?" Answer: "Presque toujours!" Question: "Was?" (What?). Answer: "Travaillé .... chose .... je ne sais pas bien." He answered eagerly in the affirmative when asked if he had worked in the Paris chalk quarries, but could give no further details.

At the autopsy we found a reddish yellow, circumscribed softening of the *left cerebral cortex in two foci:* the larger, 5.5 cm. long and 1.5 to 1.8 broad, occupied the anterior portion of the "*Gyrus angularis*" (Huxley, Ecker); the smaller, 2 cm. long and 0.6 broad, partially destroyed the "*Gyrus occipitalis II.*" *where it unites with the* "*Gyrus temporalis II.*" The depth of the foci was only 4 mm. The branches of the vessels leading to them were the seat of thrombosis. There existed no other lesions of the brain. Unfortunately, we were unable to ascertain whether the paresis of the right arm had its origin in thrombosis of the brachial artery or in the lesions of the cortex.

Our patient, therefore, was able at the best to put together words of three syllables, never one of four. He presented the typical picture of loss of memory for spoken and written words *without special complications*, which manifested itself principally by a *forgetfulness of substantives and by the markedly disjointed manner in which he combined letters and syllables in the spoken words.* The recognition and comprehension of words were not noticeably affected. This case illustrates admirably how *an ataxic impress can be stamped on the utterance of a word by the disjointed acoustic combination of the same.*

We will now describe another case, also observed by ourselves, in which the recollection of nouns and verbs had suffered seriously, and their formation from the sound and syllable elements but little, while the *comprehension of words* seemed affected. This case forms the transition to the derangements which we shall describe in the following chapter as *word-deafness* and *word-blindness.*

A gentleman of fine appearance, sixty-six years of age, with sclerosis of the arteries, and fond of good living, was in good health until five weeks before he came under our observation. At that time, while on a journey and suffering from obstinate constipation, he began to be visited by attacks of dizziness attended by unusual mental irritability and ill-humor, with headaches in the right frontal region, which at times were very severe and disturbed his rest at night. Gradually a disturbance of speech developed itself, on account of which I was consulted. He greeted me at first with some tolerably fluent sentences, and recalled a previous accidental meeting in Stuttgart. Presently, however, he came to a full stop in each new sentence as soon as he came to a noun, or used instead a wrong word (paraphasia). If he was helped over this difficulty, he was able to conclude the sentence correctly, provided he did not stumble across a verb at the end; if he did, he generally exchanged it for an inapposite one. It often seemed to me that the verb did not occur to him, simply for the reason that the first part of the sentence had vanished from his memory. His inability to find the substantives vexed him; he tried to paraphrase them, but in doing so involved himself into more and more incomprehensible combinations of phrases, and lost the thread of his sentence. He became tranquil, however, and joyfully assented when I guessed what he really wished to say. Mutilation and distortion of words occurred only to a slight extent.

Being far-sighted, he used glasses in reading, and with their help read aloud, somewhat slowly but correctly, even very long words. It was evident, however, that many words were strange to him which had been unquestionably familiar to him previously. Thoughtfully repeating one of these words twice, he remarked, "I have never met this before." In speaking the longest words he broke off in the middle, repeated the first half, and then united both halves into a well-spoken whole. He could write letters, but was unable to write words correctly. He began with wrong letters, then wrote perhaps two or three in correct sequence, and finally finished the word with a number of wrong letters. The effort of writing fatigued him greatly, and he gladly desisted from it. The movements of the tongue were free in all directions and he could whistle loudly. During hearty laughter a tolerably marked drawing of the mouth towards the right side was observable, but no drooping of the left angle of the mouth could be discovered. The limbs were free in all their movements, but the patient complained of being easily fatigued. It was discovered that he had always been left-handed; he wrote, however, with the right hand. This was weaker than the left (from want of practice), and, as has been said, was not paralyzed. He saw well with both eyes; he had been for a long time slightly deaf in the right ear; sensation in the skin and the tongue was delicate. When the patient was told to press the hand, to put out the tongue, or to close the eyes, he spoke some assenting words, "ja, ja," or "ja wohl," but did not perform the movements until they had been made before him once or oftener. He acted in this like a person who does not understand a request couched in a foreign language, and who can only be made to understand by gestures.

I saw the patient again fourteen days later. His mental and bodily strength had in the meantime markedly decreased, and a *left-sided* hemiparesis of the face and arm was unmistakably present. He used the right hand almost entirely, although he had seldom used it previously, and passed it frequently over the right side of his head, which seemed to pain him. He spoke only a few, for the most part unintelligible words, and no longer understood what was said to him. His food had to be put into his mouth, because he was no longer able to get hold of it with the spoon and fork (*apraxia*). He only ventured to walk when he was led, and walked then with short mincing steps. After this time all the faculties of the patient progressively decayed, and he died three weeks later.

At the autopsy, as the physicians kindly informed me, the *right* temporal lobe was found swollen, and affected with yellow softening in its anterior portion over an area about the size of a goose-egg, without sharp demarcation from the healthy medullary substance. At the fissure of Sylvius a portion of this lobe, about as large as a hazel-nut, was infiltrated with blood. The softening extended some centimetres downward into the head of the nucleus caudatus. The frontal lobe was intact.

At the date of the first examination in this case there was evidently *not only a derangement of association between the conception and the word in the direction from the former to the latter, but also a derangement in the direction from the latter to the former;* the *expressive* as well as the *impressive* band of fibres was affected, while the words were still properly made up of their sounds, whenever it was possible to drag them from the depths of the memory, whether this were done by the force of the idea itself or of the written or dictated words. At the same time this case shows how *the loss of nouns and verbs can impede the course of the speech, and thereby interfere with the evolution of the thoughts themselves.*

We are thus led to the question of *the relation of word-amnesia to the intellect.* We see, from the example just cited, that this defect in its severer grades renders well regulated thinking *aloud* impossible. It does not follow, however, that *silent* thought is also incompatible with it.

There are *many mental combinations that are consummated without words.* To such belong particularly many *games.* Thus, we see many aphasics, who can hardly find a single word, still play dambrett, dominoes, cards and chess with great skill. The aphasic Paquet, of whom we have repeatedly spoken, who could not even count his age upon his fingers, and whose intelligence was considerably impaired, could still play cards well, and cheated thereat with a certain shrewdness (Trousseau). It is a remarkable fact that persons who previously played well sometimes remain masters of the game, not only when they have become aphasic, but also when they have become weak-minded. Trousseau tells us that when he was Assistant Physician in the Insane Asylum at Charenton, he was often irritated to the very depth of his soul, because some half-idiotic person beat him at a game of trictrac or chess.

Mental combinations which bear upon business affairs, and can only be set in operation by the aid of speech, are at times even admirably carried out notwithstanding the existence of a high degee of ataxic aphasia. We have already cited in Chapter VII. an observation of Broadbent's, belonging in this category, which places this fact beyond doubt, and we will now quote one from Trousseau that is no less instructive.

An educated landed proprietor, who could only say "oui" with a heavy lalling tongue, and could not write at all, suffered not only from anarthria, but also from aphasia. When requested to pick out the o, u and i, of the word "oui" from a number of letters more than one ctm. in size, he succeeded in doing so, with some difficulty, but was not able to form the word "oui" out of them. He understood very well what was said to him. His intelligence was impaired, it is true, as the members of his household showed, but to so slight a degree that he was still able to aid his son with useful advice in the management of his large property. He insisted on being consulted in regard to the leases, indicated by gestures when any clause displeased him, and would not then rest content until it was changed in a manner to suit him. As a rule, the changes he sanctioned were profitable. He played cards as well as he did previously.

This patient, consequently, thought out correctly involved questions of business, although he suffered in an advanced degree from ataxic aphasia. It is not necessary to regard the decline of his mental power as a consequence of the aphasia; both derangements could be co-results of the unquestionably serious organic lesion of the brain which had caused permanent hemiplegia and paresis of the tongue. *Impairment of the intellect is almost invariably met with in connection with aphasia, but the two derangements do not run a parallel course, and hence it is advisable not to ascribe an existing mental debility* *to the aphasia, but rather to seek for the cause of both disturbances in a third, the organic lesion of the brain.*

One would think that *amnesic* aphasia, especially in its more severe forms, must offer a greater obstacle to mental combinations of the second class than the ataxic variety of the affection—that the loss of word-signs must render the thoughts mixed and confused. In fact, unless the affection be merely a light form of aphasia of recollection, it is almost always accompanied by a very pronounced diminution of the intelligence. Lordat, however, asserts, as we have already mentioned in Chap. VII., that he was able to think rationally in spite of an amnesic aphasia with entire loss of the memory and understanding for spoken and written words. As we do not consider ourselves justified in refusing all credence to Lordat's assertion, although it evoked many doubts in us, as well as in Trousseau, we are compelled to leave the answer to this question open.

It is not always easy to draw the line between the aphasia of recollection and deeper disturbances of memory. Whilst in the former we have to deal only with a disturbance of the conduction from the ideational centre to the acoustic word-centre, with a diminution consequently of the ability to draw out the word-signs from the memory through the stimulus of the thoughts, in the latter the function of the formation of signs and the acoustic word-centre itself are involved. Up to this point the conditions of amnesic aphasia can apparently be easily and clearly grasped. They become, however, more complicated and often difficult to unravel, when, as in our last-mentioned case, conduction in the direction from the acoustic word-centre to the ideational centre, *i. e.*, the comprehension of the signs, is affected; when, further, the track between the acoustic and the motor word-centres is involved in the lesion; when the intelligence is greatly impaired; finally, when, in the case of persons who know how to read and write, the optical word-centres employed in reading and writing and the bands of fibres connecting them with the ideational centre and the motor centres for spoken and written words, must be taken into consideration in the analysis of the case. We will describe some difficult cases of this character here; in the following chapters they will be more minutely investigated.

In one case, which was published by Dr. Hertz[1] in the last century, the word-images very seldom came spontaneously to the lips, and then only in an isolated manner; the words, moreover, could only be repeated from dictation, when they were pronounced several times loudly before the patient, while, on the other hand, in reading they came to him instantaneously.

An officer, about forty years of age, was attacked with apoplexy in August, 1785. The tongue, hands, and feet, were at first paralyzed. After a year he was able to walk again, and his hands also had become stronger, but a disturbance of speech remained behind. He pronounced distinctly isolated words that happened to occur to him, or that were repeated before him a number of times aloud and slowly; otherwise his discourse was an incomprehensible muttering. When a book or manuscript was put in his hand, he read easily and distinctly, and not even the smallest mistakes could be noticed. As soon as he laid aside the book, he was unable to repeat a single one of the words just read.

In the following case the restoration of the memory for words by the aid of written speech and spelling is extremely interesting.

Dr. Hun,[2] of Albany, reports the case of a farrier, who suffered from heart disease, and was seized one day with congestion of the brain, which threw him into a state of stupor that lasted several days. He then recovered consciousness and understood what was said to him, but although his tongue was freely movable, he could not find words, and had to make himself understood by signs. He understood the meaning of the words which were said to him, but could not find those necessary to give expression to his own thoughts. He could not repeat words that were pronounced before him. When Dr. Hun pronounced the word which he sought, he was much rejoiced, but his efforts to repeat it were fruitless. If Dr. Hun wrote it out for him, he was able to spell it, and could then pronounce it after a few attempts. He gradually learned by repeated practice to retain and use it. He made for himself a tablet on which the necessary words were written out, and took refuge in this during speaking. Finally, he learned to do without it. When he was unable to pronounce a word, he was also unable to write it.

We follow this up with a case in which the entire domain of expression and intelligence was deeply involved. The aphasia in this case seemed more considerable than the agraphia, and there was evidently a severe disturbance not only of an amnesic, but also of an ataxic nature. The patient was treated at first by Trousseau, and later by Charcot.[1]

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[1] *Winslow*, loc. cit.

[2] *Bastian*, loc. cit., p. 220.

Adèle Anselin was at first entirely speechless and paralyzed upon the right side. The hemiplegia persisted, but the tongue was freely movable, and subsequently she became able to speak of her own accord three words or phrases: "Maman! maman!" to call people to her; "Peux pas dire," as an answer to all questions; and when she was impatient, "Oh, malheur!" Besides this she could count up to fifteen or sixteen. She was somewhat better off in regard to writing, for which she made use of the left hand. She was able to write down several short phrases without dictation, *e. g.*, "Monsieur, je vous remercie de toutes vos bontés!" She could also write a few words and short phrases which were dictated to her or which she read, but she frequently mixed up the words, and generally ended with undecipherable hieroglyphics. She often employed the wrong pantomimic symbols. A childish behavior betrayed the fact that her intellect was affected. The autopsy disclosed numerous foci of softening in the cerebral cortex, which were secondary to inflammatory vegetations in the left heart. They occupied chiefly the left island and the border convolution of the left temporal lobe, the third right frontal convolution, and the right corpus caudatum.

In the following case, finally, there was also severe amnesic aphasia, along with serious disturbances of the understanding and of the intelligence in general. The motor centre for spoken words had not suffered, but the connection between it and the acoustic sound-centre seemed at times to be interrupted, whereby the aphasia received a more ataxic impress.

A patient, whose history was given to us by Sander,[2] could speak a number of words in a tolerably connected manner, but often missed single words, and sometimes could not tell his name, age, and occupation. Occasionally he was able to pronounce the most difficult words, *e. g.*, "Schornsteinfeger" (chimney-sweep) six times in succession. At other times he repeated incorrectly words that were pronounced before him, and did not correct the mistakes, no matter how often they were repeated to him in the proper way. He remarked occasionally, when he did not know a word, "I did know that, but I cannot say it any more!" He could only count up to twenty. When asked how much six times seven is, he answered, twenty-six. He could not repeat the alphabet; when he tried to do so he would say, a, b, c, 3, 4, 5. He could no longer read or write, and also no longer recognized individual letters. He was unable to name objects placed before him, but found them correctly when their names were mentioned to him. When he was told to look at his finger, he raised his hand, attempted to whistle, and the like. The affection ended in imbecility. The autopsy revealed an extensive focus of softening in the left hemisphere, in collections of fibres which, according to Sander, belong to those radiating from the corpus callosum.

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[1] *Font-Réaulx*, loc. cit. Obs. 33.

[2] Arch. f. Psych. Bd. II. S. 53. Fall 4.

CHAPTER XXVII.

Word-Deafness.—Word-Blindness.—Derangements of the Impressive or Perceptive Speech-track in General.—Alexia and Kindred Derangements in the Comprehension of the Symbols of Expression.—Apraxia and Aphasia.

In medical literature we find cases recorded as aphasia which should not properly be designated by this name, since the patients were still able to express their thoughts by speech and writing. They had not lost the power either of speaking or of writing; they were no longer able, however, although the hearing was perfect, to understand the words which they heard, or, although the sight was perfect, to read the written words which they saw. This morbid inability we will style, in order to have the shortest possible names at our disposition, *word-deafness* and *word-blindness* (*cæcitas et surditas verbalis*).

These defects seldom occur isolated; they are generally combined with other dysphasic derangements, with genuine loss of words of an amnesic nature, or with agraphia. We have already described such cases in which, besides aphasia and agraphia, there was impairment in a varying degree of the ability to understand the words which were heard or the writing which was read. As, however, a correct appreciation of this remarkable phenomenon is of the greatest importance for the comprehension not only of aphasia as a morbid affection, but also of the mechanism of speech in general, it is necessary to make it the subject of a special examination.

All disturbances of speech can be brought under two great classes, *according as the connection between the conception and the word is impeded in the direction from the former to the latter, or vice versâ, from the latter to the former. When the first happens, the expression suffers; when the second, the understanding.*

We call the entire tract which leads from the nerves of sense to the centre of conceptions the *impressive or perceptive;* the other, which compasses the expression of the conception, *the expressive.* What we style diction and articulation move entirely upon the expressive tract. The impressive tract serves only for the reception of words spoken or put into writing by others; hence, the ability to learn to speak and to write depends upon the integrity of this tract. From the clinical facts that will presently be investigated, the conclusion is evident that, as in the expressive tract organically different functions are called into action for the production of sounds and words, so in the impressive tract different organs are required for their reception. For the understanding for sounds and that for spoken words, or the understanding for written letters and that for written words, are not inseparably bound together, but are different things. The understanding for words can be lost, and that for sounds or letters retained. *The perception of sounds and murmurs which are individually known as vowels and consonants, and their arrangement into the acoustic word-image which becomes the symbol of this or that idea, are different functions, which are performed in different parts of the central system.* The aphasia of Lordat, of which we have already repeatedly spoken, is one of the most interesting examples of *entire inability to speak due to loss of the memory for words with complete word-deafness and word-blindness.* We will linger a little longer over his case.

"Je me trouvai privé de la valeur de tous les mots. S'il m'en restait quelques-uns, ils me devenaient presque inutiles, parceque je ne me souvenais plus de la manière dont il fallait les coordoner pour qu'ils exprimassent ma pensée." This statement, it is true, leaves it uncertain whether Lordat was entirely deprived of the word-images; but it is altogether probable that he was, since he not only could not utter a single word, but distinctly states that the words fell uncomprehended upon his ear, although his hearing was preserved and he was able to reflect as a physician and philosopher upon his condition. In the same way the treasures of writing were closed to him as with seven seals. *He could spell, it is true, but could not read.* "En perdant le souvenir de la signification des mots entendus, j'avais perdu celui de leurs signes visibles. La syntaxe avait disparu avec les mots, *l'alphabet seul m'était resté;* mais la jonction des lettres pour la formation des mots était une étude à faire. Lorsque je voulus jeter un coup d'oeil sur le livre que je lisais, quand ma maladie m'avait atteint, je me vis dans l'impossibilité d'en lire le titre.

Il m'a fallu épeler lentement la plupart des mots." He describes with deep
tion the happy moment when, after several wretched weeks, he allowed his g
wander over his library, and unexpectedly, from out of a corner the words "
pocratis opera" flashed upon him from the back of a folio. Tears burst fro
eyes. That moment marked the commencement of the improvement, which f
ended in recovery.

Another not less important observation reported by
Schmidt,[1] of Münstermaifeld, shows that word-deafness
word-blindness can accompany an amnesic derangement of sp
of an intense grade, which in this case manifested itsel
aphasia, paraphasia, mutilation and distortion of words,
agrammatismus. The words were only perceived as a *conf*
murmur, although the hearing was very delicate, and altho
the *vowels and consonants could not only be recognized*
alone, but also could with some trouble be combined into wo

A woman, twenty-five years of age, became suddenly unconscious, during
straining at stool, ten days after confinement. After consciousness returned sh
not paralyzed, but suffered from aphasia and paraphasia. She found the
with difficulty or not at all, reversed or mutilated them, said "butter" inste
"doctor," threw out letters and syllables, inserted others, used the infinitive in
of the proper mood, and conjugated irregular verbs regularly. She was th
to be deaf because at first she did not understand a single word. It was soo
covered, however, that she heard a knocking at the door or the ticking of a
as clearly as a well person, that she could distinguish between two house-cloc
the tone, etc. Words, on the other hand, as she afterwards stated, were perc
only as a confused murmur. She heard separate vowels and repeated
When a word of one syllable was spoken in the ordinary way, she did not
stand it, but when the different letters were separated distinctly from each
so that they stood forth in the pronunciation, she was able to repeat the
With words of more than one syllable it was necessary first to pronounce on
lable distinctly, then another, then the two together, or she would not under
the word. It was the same thing with reading. She studied the words very
fully, and tried to pronounce them at first separately, and then together. R
ery took place slowly. She did not understand short sentences until after the
of half a year, and then only when they were pronounced slowly and disti
Even at the last there remained some little difficulty in speaking.

Patients who suffer from word-deafness and possess at
same time the ability to express themselves in words, but

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[1] Allg. Zeitschr. f. Psychiatrie. 1871. Bd. XXVII. S. 304.

many words in the wrong places, and often distort them, leave on the minds of observers the impression that they are crazed. The observer must be on his guard to avoid falling into this error, or into the still greater error, which has also been committed, of regarding the patients as at once deaf and demented. The patients may have perfectly correct ideas, but the correct expression for them is wanting; the words, and not the thoughts, are confused. They would even understand the ideas of others if they could only understand the words. They are in the position of persons suddenly set down in the midst of a population which uses the same sounds, but different words, these striking upon their ears as an unintelligible clutter. They attempt themselves to speak this language, which perhaps they had once learned during childhood, but had almost entirely forgotten long before. They no longer, however, find the right words, and those which they find are uttered in distorted and incomprehensible shapes.

Baillarger[1] demonstrated once that a person who had been regarded as both deaf and demented, was really neither one nor the other. He concluded, from the expressive gestures with which the woman, who did not even know her own name any more, accompanied her disconnected discourse, that perfectly distinct ideas existed behind the apparent confusion. Unlike an insane person, *she betrayed a knowledge of her own condition*, and never did anything silly.

Wernicke[2] described two very instructive cases of this kind. One of the women whom he treated for this disease was considered by the attendant to be deaf.

Both women were advanced in years. On a superficial examination, one of them made the impression that she was insane, partly because her answers did not correspond to the sense of the questions, partly because she used inverted and distorted words. The meaning of her sentences, however, could be unraveled and was found to be rational; moreover, her entire behavior was always sensible. When excited she often spoke quite correctly. When she tried to write she only made up and down strokes, and was no longer able to produce letters; she had also lost the ability to read.

Gradually she learned to speak almost correctly again and to read aloud without hesitation. She also regained to some extent the power of writing; she could copy from written manuscript, but could produce in writing only a few self-chosen or dictated words.

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[1] Bull. de l'acad. de méd. T. XXX. p. 828.

[2] Der aphatische Symptomencomplex. Breslau. 1874. Fall 1 u. 2.

The other person, who also suffered from paraphasia, was at first supposed to be deaf and crazy. At the autopsy there was found, together with atrophy of all the convolutions, a pulpy, thrombotic softening of the first, and of a great part of the second left temporal convolution.

This word-deafness can only be confounded with genuine deafness when the observer contents himself with a superficial examination, since it is easy to verify the fact that the patients perceive and pay attention to calls, noises, and murmurs. Besides, the combination of aphasia with deafness is exceedingly rare; an instance of it will be found in a case of apoplexy reported by Banks.[1] This fact is striking, when we bear in mind the frequent simultaneous appearance of *aphasia and anosmia,*[2] *hemianæsthesia,*[3] *or hemiopia.*[4]

Speech-blindness occurs more frequently than speech-deafness. We must be careful, however, not to mistake the results of a *genuine hemiopia*, which usually occupies the right halves of both retinas for an inability to see and to comprehend letters and words.

According as the letters or words do or do not fall upon the remaining portion of the field of vision, the hemiopic patient can at one time see and read, at another not. When the power of recognizing words and letters is still perfect, it is easy to detect the hemiopia, but otherwise it is difficult. It is then frequently by no means easy to say how much of the alexia and paralexia is to be ascribed to the hemiopia, and how much to the more or less complete loss of the ability to apprehend letters and words. The difficulty may also be increased by the limited power of expression. The patient can perhaps still read some word or another, but can no longer pronounce it, or, when he tries to do so, produces other syllables and words. The real state of the case can then scarcely be unravelled and we stumble across the most astonishing phenomena, as witness the observations reported by Wernicke. The patients, *e. g.*, read off, while passing, the names upon the tavern-signs on the street, but are nevertheless incapable of reading letters and words with a steady gaze.

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[1] Dublin Quart. Rev. Febr., 1865.

[2] *Bonnafont*, Bull. de l'acad. de méd. T. XXX. p. 875.—*W. Ogle*, Anosmia. Med.-chir. Transact. LIII. p. 263. Compare also the case described in Chap. XXIX., p. 787, in which the outbreak of aphasia was accompanied by hallucinations of smell.

[3] We have detailed several observations of this sort, reported by *Cornil*, *Perroud*, and others.

[4] *Sander*, loc. cit. Fall 9. *Bernhardt*, Berlin. klin. Wochenschr. 1872, No. 32. *Wernicke*, loc. cit. Fall 3 u. 9.

They recognize the individual letters, but are unable to put them together to form words, or, *vice versa*, they read the words but cannot read the individual letters. A patient recognizes the word Goethe on the title of a book, but not the word Schiller on that of a neighboring one. Long words are perhaps often read wrongly only for the reason that, while the first syllable can still be properly seen, the rest cannot, and the word is then filled out arbitrarily in accordance with the idea called up by the first syllable (Bernhardt[1]).

Wernicke made a very sagacious remark in regard to the ability to comprehend written or printed characters. He called attention to the fact that this is also dependent upon the *degree of intellectual cultivation*. The uneducated man, who is but little practised in reading, can only understand the words written *with the aid of their spoken images ;* he has *to read the writing aloud* to himself, because, for the understanding of the sight-images, he requires the aid of the sound-images. The scholar glances over a page, and understands its meaning without first translating the written words into spoken words. The first will present the symptom of alexia in addition to the aphasia ; the other can understand written language, and his ability to do so presents the most striking contrast to his inability to understand spoken language. On the other hand, when he attempts to read aloud, he may be as aphasic as in spontaneous speaking.

An observation reported by van den Abeele[2] leaves little room to doubt that a complete text-blindness may exist, although the power of sight, the intellect, and the power of speech are intact.

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[1] Berliner klin. Wochenschr. 1872. Nr. 32. A patient with hemiopia, which remained after recovery from aphasia, saw, *e. g.*, in the word "zerschmettert" only the first syllable, "zer," distinctly.

[2] Bull. de l'acad. de méd. belge. Nos. 6 and 7.

A woman, forty-five years of age, was struck with apoplexy while in the enjoyment of the most blooming health. After some hours consciousness returned, but she was paralyzed upon the right side, and had pain over the left eye; her intelligence was somewhat dull, the memory weak, the speech free. Six weeks later, the paralysis and the weakness of memory and intelligence had almost disappeared. Two months after the attack she discovered that she could no longer read printing and writing. She saw the text, distinguished the forms of the letters, and could even copy the text, but was incapable of translating the words into spoken words and thoughts. She could comprehend pictures, and decipher a rebus; she understood consequently ideographic representations, but not writing. When van den Abeele published this observation, the patient had already regained the power to read some words of one and two syllables.

Broadbent[1] communicates a no less remarkable observation.

A very intelligent and energetic man, after suffering from cerebral symptoms (headache, vomiting, delirium), lost entirely the power to read printing and writing. He saw the text, but did not understand it, although he could still write easily and correctly both from dictation and spontaneously. His conversation was good and his vocabulary very large, but occasionally the names of streets, persons, and things failed him; he was not able to name on demand the simplest and most familiar objects which were held up before him. He died from an extensive hemorrhage into the left temporal lobe, with rupture into the ventricle. Two old hemorrhages were found, one in the lower border-convolution of this lobe; the other and larger one, which had produced a softening of the neighboring cerebral substance, was located in the region between the lower end of the fissure of Sylvius and the ventricle at the point where the descending horn was given off.

In this case, therefore, the conduction of the word-images to the motor centre of co-ordination for writing was still perfect, while the text-images could no longer be comprehended. In conclusion, we must make room here for a very strange observation recorded by Westphal.[2]

He exhibited at the meeting of the Society for Anthropology, a retired actor, who, besides hemiplegia, presented the phenomena of an incomplete aphasia. He was intelligent, but his memory for pieces learned by heart was impaired. He spoke fluently, but he could not find and did not understand a few words. He could write very well from dictation, but shortly after was unable to read the words he had written, and he suffered in general from complete alexia. By means of a stratagem, however, as he himself very clearly explained, he succeeded in reading the word he had written from dictation upon the tablet. He passed his finger over each letter of the written word as if he were writing it again and read it while so doing. He then made a sort of calculation, and counted off the sum of the separate letters.

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[1] Loc. cit. Case 8.

[2] Zeitschr. f. Ethnologie.

We explain this phenomenon by the theory that the intelligent man *knew how to transpose the movement-images of the written letters into acoustic sound-images*, and how to combine these into acoustic word-images. The case seems to prove, as Spamer also assumes, that there is not only a path leading from the acoustic centre of word-images to the motor centre for written words, but also one from the latter to the former. At the same time the case strengthens the theory of the existence of movement-images, which accompany the production of words by the action of the muscular apparatus.

Griesinger called attention to the fact that many aphasics, when *requested to touch some portion of the body, touch* instead some other part, *e. g.*, the nose instead of the ear, without being aware of the mistake. He ascribed it to a confounding of the movements, but it is more correct to ascribe it to a confounding of the words heard, as v. Gudden[1] thought. There is question here of a state of confusion in the *perceptive* and not in the expressive tract.

Just as the understanding for written words can be lost, so can that for *figures*. An accountant read off the sum 766 figure for figure, but he did not know what the figure 7 meant before the two 6 (Trousseau). Another aphasic, on the other hand, although he could no longer count in words, could add and subtract on paper, and even multiply and divide pretty well (Proust).

We have already spoken of a patient who had lost the understanding for *musical notes*, although he was still able to play well by ear (Finkelnburg). Inversely, Lasègue[2] saw a musician with complete aphasia and agraphia, who could readily set down in notes any melody which he heard. Another patient of Proust's was able to write music in notes, and even to compose, and understood also any melody which he heard, but was unable to play from notes.

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[1] Correspondenzblatt für Schweiz. Aerzte. Bd. I., bei der Discussion über den Vortrag Biermer's.

[2] In *Proust*, loc. cit., p. 310.

Sometimes we see patients copy *drawings*, although they cannot produce them spontaneously. Whether or not the reverse occurs we are unable to say.

Many aphasics are no longer able to distinguish or to count pieces of money; they forget the conventional social forms, and the religious customs and signs; they even confound the spoon and fork, and try to eat soup with the latter. In the last case the aphasia is combined with *apraxia—the memory for the uses of things is lost as well as the understanding for the signs by which the things are expressed.* The former condition must not be confounded with aphasia, which has to deal only with the signs by which the ideas are expressed. This mistake has been committed however. We have seen that the intellect may be perfectly preserved in cases of aphasia, but this is never the case in apraxia.

CHAPTER XXVIII.

Schematic Diagram of the Centres and Tracks of Speech.

Now that we have arrived at this point, which permits us to survey the centres and tracks of speech from the standpoint of clinical facts, which are for us equivalent to as many experimental results, we think the time has come to illustrate by means of a schematic diagram the conceptions which we have formed in regard to their connections.

Baginsky,[1] Wernicke (loc. cit.), and Spamer (loc. cit.) have preceded us in the attempt to form such schematic drawings.

The diagram of Baginsky is incorrect for two reasons: it unnecessarily establishes special centres for the memory, and directly connects the centre of conceptions not only with the centre for word-images, which is correct, but also with the motor centre of co-ordination, which we believe to be incorrect.

Wernicke made the mistake of locating the centres in particular regions of the brain. For this the localization of the elementary functions of speech is not yet ripe. His diagram, moreover, only takes into consideration spoken speech, and is difficult to understand.

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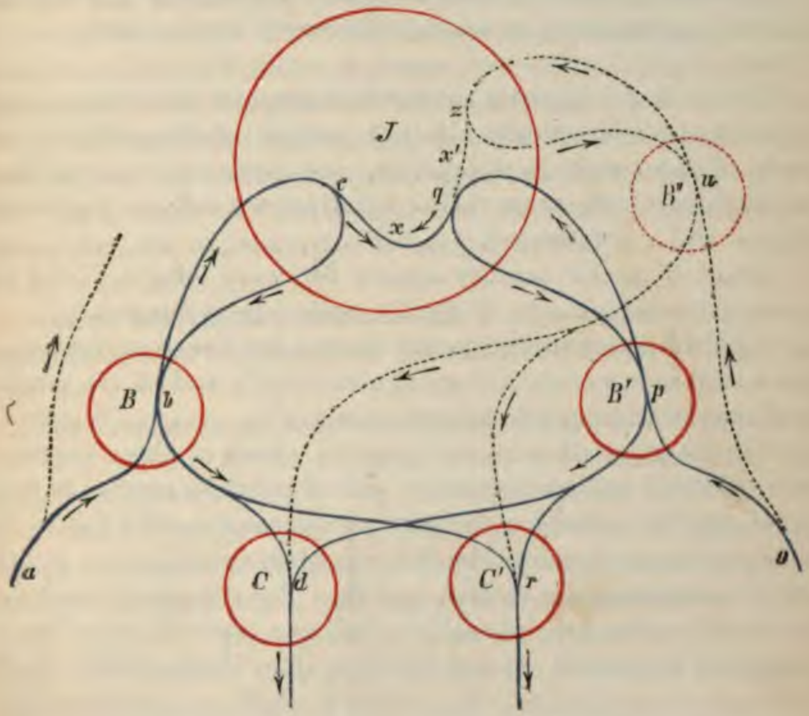
[1] Berliner klin. Wochenschr. 1871. Nos. 36 and 37.



Spamer avoided Wernicke's error of mixing up the question of localization with the task of representing the centres simply in their connections, as deduced from clinical facts. We cannot, however, agree with him in the opinion that the conceptions set the motor centres of co-ordination in action directly and without the intervention of the word-images. The results obtained from the study of amnesic aphasia speak positively against this view. In that form of aphasia the centre for conceptions and the centre for co-ordination are intact, and the word does not come to the surface, simply because the word-signs are wanting within; the recollection of the word-sign or word-image sets the word itself free by reflex action. Evidently, the excitation must take its course downward from the ideational centre through t same places of development, when a word is to be uttered, th it passed through on its way to the ideational centre, when t word was spoken by another person and perceived by the subje

We believe that our diagram gains not only in probabilit but also in clearness, by taking this theory into account.

The circle *J* designates the ideational centre or centre of cc ceptions, in other words all that portion of the cellular n work of the cortex in which ideas are produced as a result impressions of the most varied description made on the sen (object- and word-images).

B and *B'* are the sensory centres for word-images, *B* for t acoustic (sound-images), *B'* for the optical (text-images).

C and *C'* designate the motor centres for the co-ordination the sound-movements into spoken-words (*C*), and of the strol produced in writing into written-words (*C'*).

a Is the acoustic nerve, *o* the optic. Each of these nerves seen to divide into two branches, one of which, however, is in cated only by points for the sake of clearness.—*a b c b d* is t collective acoustic motor track for spoken speech, *o p q p r* t optic motor track for written speech. The adjoining punctat lines and circles are intended to indicate that still other tra lead from the nerves of sense through other image-centres to t centre for conceptions; the acoustic nerve, *e. g.*, also conv melodies, and delivers musical ideas and the sound attributes object-images (song: the nightingale), the optic nerve bri gesture-images and physiognomic attributes (grimaces: t monkey). For the sake of clearness we leave out the tracks the other nerves of sense, *e. g.*, the nerve of taste, and the mo centres of co-ordination for all other voluntary expressions, e cept spoken and written words; the centre for pantomime is a left out.

a b d is the track for the imitative speech of children parrots who repeat uncomprehended words; *o p r* is the tra for the copying of uncomprehended words; *c b d* is the track the utterance of the conceptions in spoken words; *q p r* for t writing down of the thoughts. The track *c x q* forms the conn tion between sound-images and text-images in the centre of conceptions, and renders feasible the change of spoken-signs into written-signs through the intervention of the thoughts.

b r and *p d* are the tracks between the centre for spoken-images and the motor centre for writing on the one side, and between the centre for written-images and the motor centre for speech on the other side. A person who writes down an uncomprehended word that he has heard, uses the track *a b r;* one who reads off aloud an uncomprehended written word, uses the track *o p d.* In writing down dictated words that are understood, the longer track *a b c b r* is employed; in reading aloud written words that are understood, the track *o p q p d.*

Let us now examine with the aid of the diagram the phenomena of deaf-mutism and of the different dysphasic derangements.

The *deaf-mute* can never use the tracks *a b c b d* and *a b c b r;* they remain closed for him, because the acoustic entrance gate remains unopened. On the other hand, he can arrive at *C′* and *C* through *o;* he can thus learn to write and speak. He learns to transcribe without conceptive comprehension of the text by the path *o p r*, with conceptive comprehension by the path *o p q p r.* This, however, does not suffice to unlock and drill the centre *C.* For this purpose he must erect for himself an optical image-centre *B″* for the sounds and words seen coming from the mouth of the speaker, and construct paths from *u* to *z*, and back through *u* to *d.* This centre *B″* acts vicariously for *B*, and the path *u d* for the path *b d.* There are now no further difficulties about making use of the track *q p d*, since the transmutation of mimic sound-images into text-images, and *vice versa*, become possible through the track *x′.* When the deaf-mute transcribes the words which he reads off from the mouth of others, he makes use of the track *o u r.*

In *complete ataxic aphasia*, *C* is no longer available, and hence the tracks *b d* and *p d* are also unserviceable. Spontaneous speech, repetition of words uttered by others, and the reading aloud of text, are impossible. The track *a b c*, for the comprehension of words heard, is preserved, as is also the track *c b*, by means of which the conception brings the sound-image in recollection. If *C′* is not damaged simultaneously with *C*—whi happens but rarely, undoubtedly because the motor centres co-ordination for spoken- and written-words lie very near togeth —then the patient can still express his thoughts in writing, co what he reads, and transcribe from dictation.

If the track *b d* becomes impassable, without the centre becoming functionally impotent, the track *p d* can still officiat which it cannot do in ataxic aphasia. The patient cannot utt a single word spontaneously, but can read aloud what is writte —The above-cited observation of Hertz is open to this interpret tion; but, since in this case the track *c b d* was undoubtedly n entirely interrupted, we can also assume that the excitation the track *o p q* transmitted itself by the path *q x c* in *J* to *c b* and supplied the force still lacking to overcome the resistance *c b d*.

In the *amnesic aphasia*, which we styled simple *aphasia recollection*, the track *c b* is only temporarily blocked up. Wh cannot be accomplished, however, through the excitation starti from *J*, can be accomplished by the excitation admitted at and produced by the dictation of the words. The word-ima now enters the recollection in *B*, the reflex bow *a b d* can offi ate, and the bow *b c b* is re-established. The succor can arri through *o* as well as through *a*. In reading, the text-image formed in *B′*, which either renders the utterance of the unco prehended word possible through the path *p d* by reflex actio or, and this is the usual way, the image is perceived in *J*, unit with the idea, and supplies to this the force requisite to open t path *c b*.

In *aphasia, with deeper derangements of memory*, the centr *B* and *B′* are in disorder, and the tracks which lead to them a from them are obstructed in a varying degree or render entirely unfit for use.

In cases of *complete amnesic aphasia and agraphia* (Lorda in consequence of the destruction of *B* and *B′*, the collecti impressive and expressive tracks must be thrown out of use, a *J* be entirely isolated.

In the cases of *word-deafness with paraphasia*, the tra *a b c* suffers, while the track *c b* still permits conduction to the more or less weakened and disordered centre *B*, and the track *b d* is intact.

In *text-blindness* and inability to transcribe the thoughts with retention of the ability to copy writing without comprehending it (van den Abeele), the track *p q p* is obstructed, the track *o p r* retained. In text-blindness with retention of the ability to write spontaneously and from dictation (Broadbent), only the track *p q* is obstructed, the tracks *q p r* and *a b r* are preserved.

Finally, the case observed by Westphal seems to prove that there is not only a track leading from *b* to *r*, but also one from *r* to *b*.

CHAPTER XXIX.

Paraphasia.—Importance of Attention for Correct Speech.—Paraphasia due to Absence of Mind, and Choreic Paraphasia.—Paragraphia.—Paralexia.—Paramimia.

Under the name *paraphasia* we describe that disturbance of speech in which *the connection of the ideas with their word-pictures has become so disordered that, instead of the appropriate terms, words having a different meaning, or altogether strange and unintelligible expressions, are enunciated.*

The disturbance of speech which is occasioned by impeded utterance or stammering, in which the correct words are used, although in distorted and confusing shapes, is not included under paraphasia. Neither is this name appropriate for ataxic *aphasia* in which the patient, like an automaton, answers all questions with the same stereotyped words or fragments of words.

The differentiation of *paraphasia from amnesic aphasia and syllable-stumbling*, affections with which it is sometimes but not necessarily combined, is more difficult. It might be thought that in paraphasia the faculty of recalling the correct word is always impaired, since instead of it a wrong one is employed; but this is not necessarily the case. In fact, the correct word seems very often to rise up in the memory either entirely or in part, but immediately, before it is spoken, it is crowded aside by some other word. Paraphasia may also exist when the syllabic construction of the words used is perfect. Words that are perfect in construction may be substituted for others with which they may not possess the slightest similarity in sound. Frequently the flow of speech is smooth and continuous, but the combinations of words are meaningless, and give confused and entirely unintelligible expression to the thoughts of the speaker. This affection, the so-called *choreic paraphasia*, in which the entire vocabulary of the individual is wrenched from its normal connection with the intellectorium, can be easily differentiated from syllable-stumbling, provided the words employed are borrowed in their correct forms from the mother-tongue or from foreign languages. The difficulties begin, and syllable-stumbling and paraphasia become closely related, only when the perplexing words are formed by the combination of sounds and syllables borrowed from different words. We speak of *syllable-stumbling*, when the words whose sounds and syllables are intermingled *follow each other in the sentence;* of *paraphasia*, when the words *do not properly belong together in the sentence*, but have risen up in the memory in consequence of some resemblance in meaning or sound.

There is a form of paraphasia which does not overstep *physiological limits.* It is not an uncommon thing for sound, healthy men to make mistakes in speaking, of which they may or may not be conscious. These mistakes are due to *absence of mind.* The same cause—*want of attention*—which may develop into a state of *intellectual confusion*, is also usually at the bottom of the *pathological* paraphasia.

By *attention* we understand that state of mind in the individual in which he takes cognizance of the processes that have been aroused in the perceptive tract by external or internal agencies. It is possible thereunder to conceive of an excitation which, taking its origin in the ideational centre, would be conducted, not only along the motor nerves to the accessory muscles of the apparatus of speech, hearing, taste, etc., but also centrifugally along the sensory nerves to the external organs of sense. It is, at all events, certain that by means of attention we exercise an influence on the motor and sensory central apparatus of language which is indispensable for correctness of speech. When the attention flags, inappropriate words are very often used, and such liberties may be taken with the accentuation, the rapidity of speech, and the literal and syllabic enunciation, as to render the speaker unintelligible.

This attention to what is being said leads to favorable results only under certain *conditions :*

1. It must be accompanied by a *feeling of self-confidence.* Timid individuals are more liable to make mistakes in speaking than self-sufficient and forward persons.

2. The thoughts must be fixed on *the idea to be expressed,* and not allowed to wander off to matters not connected with the subject. Men who are easily distracted from a train of thought by external impressions, or who do not curb the rapid flow of ideas aroused by associations, are said to be *absent-minded.* They are constantly in danger of losing the thread of the discourse, and of using words which are foreign to the subject.

3. The attention, while speaking, must be divided between the thoughts, the syntax, and the words, *each receiving the modicum that it requires.* When the individual words engage the attention too exclusively, the thought and the construction of the sentence easily slip from the memory, and the speaker is liable to become entangled in a maze of words and phrases. Here we have at the same time *akataphasia* and *paraphasia.* In pathological conditions, only very slight disturbances of the circulation and nutrition of the brain are often all that is necessary to weaken the power of attention and occasion paraphasia. Persons who are suffering from fever or from hunger, or who have been stunned, easily become confused in speaking. This is still more liable to be the case in connection with all those circumscribed or diffuse, macroscopic, organic lesions of the brain which are attended by obscuration of the intellect and impairment of the bodily powers in general, and of the power of speech in particular. The *thoughts are confused, and the words are mixed up in a most perplexing way.*

It is especially words expressing things that are in daily association, that are liable to be exchanged for one another; thus, stick and hat, knife and fork, salt and pepper, etc., etc.; or the words for ideas that are included in the thought to be expressed. Thus, a physician suffering from fever wishes the door to be opened to let his dog out, and calls out, "Open the dog." Or the word which correctly expresses the idea rises up in the memory, but, before it can be enunciated, another word resembling it in sound or in meaning is suggested and spoken in place of the correct one. Instead of butter, the word mother, or doctor (see p. 772), or cheese, is used; instead of pamphlet, camphor; instead of drinking vessel, chamber-utensil (Trinkgeschirr, Nachtgeschirr—Crichton)

In this connection a case observed by Trousseau is very instructive. A woman was affected with aphasia, after recovering from an erysipelas of the scalp, that had been accompanied by cerebral symptoms. She learned first to repeat words spoken by others, and then for several months daily wrote down words in a notebook, in order to strengthen her memory. It was interesting so see how one word led to another. Sometimes the first, sometimes the second syllable, often also the rhyme, or a very slight resemblance in meaning, gave the key to the following word. Thus, "chat, chapeau, peau, manchon, main, manche, robe, jupon, rose, bouquet, bouquetière, cimetière, bière, mousse," etc., etc.

In many cases the transposition does not involve entire words, but particular syllables or sounds are borrowed from words presenting similarities in meaning or sound; or an exchange of syllables takes place between words that are connected together by the association of ideas, or that belong together in the same sentence. In the last case the paraphasia becomes associated with syllable-stumbling.

An absent-minded professor, to the great amusement of his hearers, spoke of "the two great chemists Mitschich and Liederlich," meaning Liebig and Mitscherlich. Our unfortunate aphasic patient, Senn, called a "Gabel" (fork) "Gasser," because, while thinking of "Gabel," the word "Messer" (a knife) also came into his mind.

Alliteration is particularly dangerous for those who do not possess a thorough command of the power of speech.

Even the best runners sometimes stumble. We once heard an after-dinner orator, who was renowned for his readiness and skill in speaking, make a mortifying mistake. He was addressing a large and learned society on the occasion of an anniversary dinner, and, indulging in too rapid a flow of speech, he welcomed the members to the "festlichen Fressfreude" instead of to the "fröhlichen Festfeier."

In the slighter forms of paraphasia the thread connecting word and idea is only occasionally entangled, but in the severe forms, which we call *choreic paraphasia*, the entire web of connecting threads is involved in inextricable confusion. When, as is almost invariably the case, the connection between the idea and its written picture is also disordered or entirely broken off, there is no key left which can unlock the meaning of this *medley of words* or *verbal delirium*. A case of this sort came under our observation many years ago.

A pedagogue, sixty-five years of age, with rigid arteries, exerted himself beyond his strength. In August, 1870, on returning from a walk that had been enlivened by animated conversation, he sat down at his desk, but soon afterwards sprang up and cried out: "I smell sulphur! don't you smell anything? I am struck with apoplexy!" He then vomited; for several days subsequently he was affected with *verbal amnesia*, but there was at no time any paralysis. He recovered and resumed his herculean intellectual labors. In autumn he had another attack of vertigo without paralysis, which was followed by a *paraphasia* that rapidly attained very unusual intensity. He addressed long speeches to the persons about him, but was nevertheless unable to express even his simplest wants in intelligible terms. On one occasion, for instance, an entire day was expended in a fruitless endeavor to make his sister, who kept house for him, understand that he wanted her to bring back a bed coverlet that she had taken away. He finally succeeded in making her understand his desire, by repeatedly striking on the bed. His actions meanwhile gave no indications of any marked disturbance of intelligence. He still understood what was said to him, and was greatly pleased when any one divined the meaning of what he said himself. I only saw him once, on the 23d of February, 1871. He advanced courteously to meet me when he entered the room, and at once addressed me in a long, fluent speech, of which I comprehended absolutely nothing at all. It was made up of German words grouped together in an entirely meaningless way, but it was evident, from his countenance and his gestures, that he was endeavoring to describe his condition. His demeanor was like that of an animated speaker lecturing from a professor's chair. A foreigner unacquainted with our language would unquestionably have supposed that an intelligent, cultivated man was delivering a well-digested speech. He did not seem himself to notice that his remarks were unintelligible. I examined him, found no paralysis, and then spoke a few encouraging and comforting words, which had the effect of making him cheerful and happy. It seemed to me that he actually understood the meaning of my words, and did not merely guess at them from my countenance. In the early part of spring, as I afterwards learned, his intelligence rapidly waned, but he nevertheless continued for some time to play a good game of cards every evening. He always employed a considerable portion of his time in writing, and I have now in my possession a book in which he had written some notes. The written words and signs begin as follows: "Insera—Ihrewaesch—Im Jahre—Wascho—Jnd—Leib—Kal—7. 8 Juan. 6. Nachsidhidlig unsersehsch t im 1 weiss diesebelse heft, Sechal forsich Br schaf 5 bis mich hoff. Ich habe," etc. Gradually he began to confound material objects; he would try, for instance, to eat soup with a fork. He died, a corporal and intellectual ruin, on April 1, 1871.

One of the most remarkable of the recorded cases of disturbance of speech in general, and choreic paraphasia in particular, has been reported by Dr. Osborn.[1] The connecting web which unites the ideas with spoken words was in a state of inextricable entanglement, while that which unites the ideas with written words was unaffected, and the intelligence was apparently perfect. It was possible to understand what the patient wrote, but not what he said. He himself understood what was said to him, and what he read; but when he tried to read aloud, he gave utterance to a string of curious words, some of which were English, and some were not; a portion of the latter were borrowed from foreign languages, but others belonged to no language under the sun.

The patient was a literary, thoroughly educated man, and had made himself master of three languages. After an attack of apoplexy, he was no longer able to speak in an intelligible manner, although he was not paralyzed and could enunciate with great fluency a great number of words. Persons who did not know him, supposed he was speaking some foreign language, so fluently did syllable after syllable, and word after word issue from his mouth. He understood all that was said to him and also what was written, and was himself able to express his thoughts rapidly in writing, very rarely using incorrect words. The power of calculating was unimpaired. He could not repeat words pronounced before him, with the exception of a few monosyllables, and neither could he repeat the letters k, q, u, v, w, x, and z, although in speaking he was able to use these sounds with the others. When he was told to read aloud, he uttered a farrago of words that had no meaning, and did not correspond with the written or printed words. For instance, he read the sentence: "It shall be in the power of the College to examine or not examine any Licentiate previous to his admission to a Fellowship, as they shall think fit," as follows: "An the be what in the temother of the trothotodoo to majorum or that emidrate eni enikrastrai mestreit to ketra totombreidei to ra fromtreido as the kekritest." Another time, in reading the same sentence, he employed different words, but his gibberish was equally unintelligible.

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[1] Dublin Quarterly Journal of Medical Sciences for 1833. Vol. IV. p. 157.

Not only can a sound and healthy person *make mistakes* in speaking, but he can also, and even more easily, *make mistakes* in writing. While writing, the thoughts are more easily distracted, and consequently there is a greater liability to use letters, syllables, and entire words incorrectly. Words presenting resemblances in orthography, as well as those allied in meaning or in sound, are liable to be employed for one another. The same is true of invalids, in whom the functional and organic lesions of the brain are more readily betrayed in the writing than in the speech. *Morbid paragraphia*, like morbid paraphasia, presents itself in mild and in severe forms.

An excellent example of paragraphia by transposition of the words in the sentence is found in the following certificate, which was given by an absent-minded professor to a student who had attended his lectures on inorganic chemistry: "Mr. Schmidt has attended my remarkable lectures on chemistry with inorganic assiduity."

The case reported by Osborn, in which the paraphasia was almost entirely uncomplicated with paragraphia, has no parallel in medical literature. In every form of dysphasia the power of writing is, as a rule, even more impaired than the power of speaking. A few such cases, which apparently might be classed with equal reason under the head of paragraphia, are here given.

Hughlings Jackson[1] saw a woman five weeks after an apoplectic attack, which had been followed by right-sided hemiplegia, and by aphasia that for a week had been complete. The hemiplegia had entirely disappeared at the time of his visit, and the aphasia had diminished so greatly that he almost overlooked it. When, however, the patient attempted to write her name, she wrote, "Sunnil, Siclaa, Satreni," and her address she gave as follows: "Sunesr nut to mer tinn-lain."

The same author communicates the letter of a gentleman who suffered from moderate verbal amnesia and aphasia, that were consecutive to epileptic attacks. It contains a number of incorrectly written or altogether inapposite words, but it is still possible to ascertain from it the meaning of the writer. The intellect of this gentleman was not impaired. In copying he made but few mistakes. When reading aloud he spoke almost all dissyllabic and polysyllabic words incorrectly.

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[1] British and Foreign Medico-chirurgical Review. Jan. 1869. p. 231.

Bastian, from whose paper we have taken these
of his own. The patient, whose intellect was un
power of attention while speaking, and would then
ing, monotonous manner. The final syllables of th
Written words were distorted in a similar manner,
For instance, instead of "Royal naval medical offic
he wrote "Roydndendd navendendd oforendendd
we get a glimpse of something more than paraphasi
bly an instance of *dyslogical paragraphia.*

Ferber[1] had under observation a woman, six
afflicted with a paragraphia, that had persisted af
the right side and aphasia, both due to an apoplectic
instead of "grüssen," "schreigen" instead of "s
"mutter," "omdern" instead of "modern," and in
of a ch.

In *paralexia* incorrect words are utt
transposing the syllables of the printe
for them other words resembling them
orthography.

In a case reported by Jougla,[2] the patient, who
incoherence of ideas complicated by paraphasia, chang
He could still spell out the words correctly, but wh
off-hand, he gave utterance to nondescript words.
mentaires" and "établissements," he read "amouli

A patient of Bouillaud's[3] read instead of "pro
St. Paul," probably because her thoughts were dw
Paul, which had fallen on the preceding day.

The term *paramimia* is used to de
which the patient makes an affirmative
he wishes to answer in the negative,
negative motion when he wishes to an
We have already spoken of these cases.

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[1] Berliner klin. Wochenschr. 1869. No. 10. See al
ber (from *Naunyn's* clinic), Berliner klin. Wochenschr.

[2] Gazette des hôpitaux. 1872. No. 149.

Bulletin de l'académie de médecine. T. XXX. p.

CHAPTER XXX.

Syntactic Disturbances of Speech.—Agrammatismus and Akataphasia.

Steinthal[1] justly insists on the necessity of distinguishing the *syntactic disturbances of speech* from the *faulty use of words*. He proposes to call the former *akataphasia*, a name partially borrowed from Aristotle.[2]

In the *syntactic disturbances* the power of giving expression to the *thoughts* is impaired. The thoughts are expressed by means of *sentences*. Something more than mere memory for words is required for this purpose, since, "unlike words, sentences are not stored up in the memory ready for use."[3] A foreign language cannot be acquired from a dictionary alone. The ability to speak presupposes the power to give articulate expression to all the delicate shades, and more or less intricate windings, of the thought which sweeps through the brain. The most learned man is sometimes totally unable to communicate his knowledge to others.

For the expression of the developing thoughts we possess two linguistic aids, viz., the *inflection of words and their arrangement in the sentence*, or *grammar and syntax in its narrower sense; syntax in its widest application* includes both.

The *inflection of words* is not a property common to all languages; the Chinese, for instance, like the sign-languages, has no real grammar, but only a syntax. That extraordinary language has not yet made any advances beyond the radical words; the same word is a verb, a substantive, an adjective, an adverb, or a preposition, according to its position in the sentence. An individual word represents an abstract idea, to which a concrete limitation is only given by a varying intonation, or by combination with a word representing another idea. The language has neither declensions nor conjugations. In this respect the English language bears some resemblance to the Chinese. Sprung originally from languages that were rich in grammatical inflections, the English has rejected most of these and turned over to syntax the functions which were formerly performed by grammar. The prattle of children, too, presents a certain analogy to the language of the Chinese, since the meaning of their words only becomes sharply defined by their relative position to one another, or by the intonation and accompanying gesture. Moreover, even in our (the German) language, which is so exuberant in inflections, a change in the position of words often produces an entire change in their meaning: "Taschengeld" and "Geldtaschen," "ein Glas Wasser" and "ein Wasserglas" are very different things.

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[1] Loc. cit., p. 479.

[2] De interpret. C. 4.

[3] Zeitschr. f. Völkerpsychol. Bd. 1. S. 142.

Von der Gabelenz[1] has briefly formulated the supreme *psychological principle* of speaking, as follows: In thinking, the *psychological subject*, *i. e.*, the subject which is to be thought on or of, precedes the *psychological object*, *i. e.*, that which is to be thought of the subject. In all languages this principle is generally followed in the arrangement of words and sentences; as a rule, the grammatical terms corresponding to the psychological subject, precede those that correspond to the psychological object.

It is in the *sign-language* that the psychological principle of the evolution of thought is most perfectly reflected. Its syntax has been termed the *natural*, in contradistinction to the more artificial and greatly varying syntaxes of the grammatical spoken-languages.[2]

Deaf-mutes in all countries dispose the sign-equivalents, for the different ideas which they wish to express, always in the same order; the construction of their sentences is entirely independent of the syntax of their articulating countrymen. They say "horse black" instead of "black horse;" "hat black bring" instead of "bring a black hat;" "hungry, to me bread give" instead of "I am hungry, give me bread." The mute places first whatever is to him the most important, and leaves out entirely whatever seems to him superfluous (Schmalz.[3]). Or, as Scott[1] expresses it: "The subject comes before the attribute, the object before the action, the qualifier usually, but not necessarily, after the qualified."

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[1] Ideen einer vergleichenden Syntax. Zeitschr. f. Völkerpsychol. Bd. 6. 1869. S. 376.

[2] *Tylor*, Forschungen über die Urgeschichte der Menschheit. Leipzig. S. 30 u. f.

[3] Ueber die Taubstummen. Dresden und Leipzig, 1848. S. 274.

Inflection emanated from the combination of words, which led to the fusion of two or more to form a new one, representing a modification of the original meaning of the principal root. It led also to various articulate changes in the component parts of a word, which were effected in accordance with settled rules or laws. In this way cases, tenses, adjectives, adverbs, etc., were produced, and it thus became possible to give rapid and precise expression to the most dissimilar conditions of time and space, and to the most diverse relations of things to one another. The wonderful process of the grammatical development of a language extended over centuries, and was swayed by influences which, at the present time, are only partially accessible to investigation. The individual ability to *speak with grammatical correctness* is, however, the product of careful and toilsome training, for children do not easily learn to inflect, to decline, and to conjugate correctly; the uneducated man sins constantly against the rules to which the educated man adheres.

The *grammatical faults of speech* that are due to *imperfect education*, must not be confounded with the mistakes made by intellectually sound and educated persons, in consequence *of a bad habit, or of a desire for originality, or of absurd fancies.* Still less should they be confounded with those which arise from *disease*, and which we designate by the term *dysgrammatical derangements* or *morbid agrammatismus.* We frequently meet with the last as a complication of amnesic aphasia and paraphasia, and several examples of it in this connection have already been detailed. They are usually accompanied by grave disturbances of the intellect, particularly by *weakness of the intellectual powers*, or they are the expression of an *insane caprice.*

In this category belongs also an anomaly which is very frequently observed in the speech of imbeciles and lunatics. It consists in this, that the subjects do not decline or conjugate the words, but make use only of the indefinite substantive and the infinitive, and perhaps of the past participle also. They prefer the weak to the strong inflection, leave out the articles, conjunctions, and auxiliary verbs, omit the prepositions or use them incorrectly, and employ nouns instead of pronouns. They speak of themselves as "father," "mother," "Charles," etc.; instead of the pronouns "you," "he," "they," they employ the proper names or the unqualified substantives, "man," "woman," "sister," "doctor," etc. Sometimes they place the adjective after the noun, or omit the adverbial inflection. The result is to give a childish and helpless character to the speech.

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[1] The Deaf and Dumb, p. 53.

Sometimes these grammatical mistakes are made *consciously*, and are the creations of caprice and temper, but more generally they constitute the *automatic*, articulate expressions of *unconscious psychological processes.* The same holds true for the *syntactic derangements in the narrower sense of the term.*

In speaking, only the shortest sentences can be embraced in a single conscious glance. When we begin longer sentences, we know only what we wish to say; the fundamental thought or the theme of the sentence alone rises up clearly before us, while the *syntactic development of this thought in words* takes place in a *semi-automatic* manner, through the agency of the mechanism of speech. The more a speaker accustoms language to obey him, with all the force and subtlety that the spirit of the people has stored up in it during the lapse of countless generations, that is to say, the more it supports him with all its autonomic power, so much the more does he command it. The notion that the thought which the speaker is developing is the only governing power in the sentence, is incorrect; the words and the grammatical and syntactic laws are forces with which the thought must reckon. The speaker must bear in mind the points on which the sentence, as well as those on which the thought turns. Provided he does not lose sight of these, it is unnecessary for him to retain a conscious recollection of all the parts of the sentence. In a long sentence he may lose sight of the commencement, of individual words, and of entire incidental phrases, but will still be able to conduct the sentence correctly to its termination. When conscious memory fails him in consequence of its natural limitations, he is aided by a power which, working with instinctive certainty, groups the words of the sentence about the essential and pivot-words, and, provided the correct form of the sentence has been adopted at the outset, retains this form to the end.

One who is but little practised in extemporaneous delivery, succeeds much more readily in evolving artistic sentences in *writing* than in speaking. In the former, the eyes assist the mind, by means of retrospective glances at the preceding parts of the sentence. Many men who prove but dull conversationalists in a parlor, understand how to polish and mount their thoughts like sparkling diamonds, when seated at the writing-table in their studies.

The *correct diction of a sentence* in the grammatical languages *presupposes three things*, and when any one of them is wanting, *akataphasia* results.

1. *Unbroken flow of words.*

The aphasic person, who fails to recall perhaps only a single word, stops in the middle of the sentence, and, under the most favorable circumstances, finds himself forced to have recourse to a paraphrase, which, as a rule, proves distracting both to himself and to the listener. If the aphasia be marked, and a large number of words be forgotten, a correct construction of the sentence is impossible. In the slighter grades of paraphasia, the form of the sentence suffers less than its meaning, but, in the choreic form of the affection, all order and intelligibility are hopelessly lost. The following example of *akataphasia due essentially to severe aphasia*, is borrowed from Steinthal.

A man, who was suffering from aphasia and paraphasia, remarked while his eyes were being examined: "Das eine Auge — Auge ist immer — Thränen — thränig gewesen (One eye — eye has always — tears — been watery). Ich kann gar nicht — früher konnt' ich — besonders — — natürlicher Weise — mit den Jahren kleine Stiefe — Strippe — Schrift — die Brille." The second sentence, which is greatly mutilated, might be completed as follows: "Ich kann gar nicht *sehen*, früher konnt' ich *lesen*, besonders *grosse Schrift*, natürlicher Weise *musste ich* mit den Jahren kleine Schrift *durch* die Brille *lesen*." (I cannot see at all. Formerly I could read, especially large print, but, naturally, as I advanced in years I had to use glasses for small print). The words in italics are those which the patient left out.

2. *Perfect grammatical diction.*

Languages vary very greatly in their grammatical structure, and nothing can be more interesting than a comparative study of the same. We cannot, however, stop to discuss this subject in detail at the present time.

The Chinese language has no grammar, but operates only syntactically with uninflected radical words—*radical stage* in the development of language. The Aryan or Indo-Germanic and Semitic languages take the roots themselves and remodel them, with a view to the closer limitation of the thoughts—*inflectional stage.* Midway between these two classes stand the Turanian or Finnish-Tartaric languages, in which the roots are fused together to form new and complete words, in such a way, however, that the principal root, which contains the fundamental idea, remains unaltered, and only the secondary elements undergo changes—*agglutinative stage.* This fusion takes place in accordance with a fixed law. The secondary elements, which modify the fundamental idea, are dovetailed in between the primary elements, by which they are held together. In the American languages, which resemble the Tartaric, these compound words are even susceptible of declension and conjugation.

As an example of *grammatical akataphasia*, we may adduce the following remark of a feeble-minded girl: "Toni taken flowers, nurse come, Toni thrashed." The patient no longer made use of articles, pronouns, or auxiliary verbs; she conjugated, as children do, strong verbs with a weak inflection.

3. *Correct arrangement of words.*

In the Chinese language, in consequence of the want of a grammar, every deviation from the regular word-sequence must lead to misunderstandings. In German the scope is freer, but not unlimited. Strictly speaking, even in German every change in the order of the words leads to a modification of the meaning which they are intended to express; this modification, however, is often so slight that it is a matter of indifference how the words are arranged. It is only when unwarrantable changes in the regular word-sequence are made, that the sentence is liable to be misunderstood, or becomes unintelligible. Thus, to the question, "Was ist dies?" we may answer, "Weisser Zucker," or, "Es ist weisser Zucker," or, "Zucker ist es, weisser," or, "Es ist Zucker, weisser." But when any one says, "Weisser ist es Zucker," he commits a grammatical fault; he is either unacquainted with the language, or there is reason to suspect that he is not in his right senses.

Gogol[1] reports an exquisite example of akataphasia, in which the grammatical formation and the syntactical construction of sentences were greatly affected, while the patient, although his intellect was impaired, lacked neither words nor ideas. He suffered from intellectual confusion and verbal amnesia, but the grammatical and syntactical akataphasia overshadowed both these disturbances.

A young man in Breslau, who had fought through the campaign of 1866, was thrown from a wagon in 1869, and received a severe injury of the brain. At the autopsy in 1872, numerous ochre-colored spots of softening were found scattered through the cortex of both hemispheres of the cerebrum and in the cerebellum, along with traces of a former meningitis located over the two anterior lobes. After the accident, when speaking of his army life, he said: "Da war achtzehnhundert drei und sechzig Kürassier numero 1 hier in Breslau musste ich haben gewesen." That is no sentence; the following is a little better: "Und von achtzehnhundert sechs und sechzig da kommts die Preusse, auch das Oestreich gewesen, die schützen von das Oestreich." In speaking of the time when he went to school, he said: "Ich bin gewesen als Schüler — nein, ich auf das Schüler — und ich musste arbeit auf das Vater und Mutter." The patient substituted "auf" for most of the connecting words. "Da haut ich wieder arbeiten auf meines Mutter und arbeiten auf meines Vater." When asked to give an account of his accident, he said: "Ich habe 1869 als Kutscher gewesen und als Kutsche habe welche mir als ich diene haben es mir meine Pferde als Kutscher bei dem der Diener gewesen. Und ich musste es runter auf meine Kutscher und haben sie meine Eisen und haben sie Pferde auf mein Fuss und meine und da haben sie das Eisen auf meine Kopf und das Blut ist fort."

This case is also important in another connection. The patient, as is evident from what precedes, still had many words at his command, but could not always find the expressions for objects that were known to him. He could read few words correctly, but nevertheless wrote down correctly words and figures that were dictated to him, reckoned accurately, was polite, saluted courteously, and knew the sign of the cross. On the other hand, he urinated in the wash-basin, bit a morsel out of a cake of soap, and did other things of a like character, which proved that he confounded material objects with one another. He performed preposterous acts, or, in other words, suffered from apraxia. *It is evident that the misapprehen-*

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[1] Ein Beitrag zur Lehre von der Aphasie. Inaugural-dissert. Breslau, 1873.

sion of material objects, which forms the basis of apraxia, was in this case mor
nounced than the misapprehension of the articulate expressions of thought.

We may also refer here to a case reported by Forbes W
low,[1] in which the chief affection seemed to be an akataph
an inability to place words in their correct order in a sente
If the words uttered by the patient were written down, and
arranged in order, it became possible to decipher his meanin

A gentleman was seized with an attack which presented as much resembla
epilepsy as it did to apoplexy, and for two days his life was in danger. Wh
recovered consciousness, he was unable to express his wishes in an intelligible
ner. He articulated the words correctly, but mixed them up so that it was i
sible to understand him, unless his words were first written down and then co
nated. This condition lasted, with short interruptions, for fourteen days;
accompanied by severe pains in the occipital region. The patient gradually
ered under the use of venesection and derivatives.

CHAPTER XXXI.

Aphasia as a Disease, and its Manifold Causes.—Functional Aphasia.—Hys
Speechlessness.—Congenital Aphasia.—Prognosis and Treatment of Aph

The *clinical picture of* "*aphasia*" has been built up
the dysphasic derangements that we have thus far been de
ing. Its varying *manifestations*, *somatic and psychical co*
cations, *causes*, *prognosis and treatment*, have been in
gated and described. We regard this standpoint as fairly
quered. We can see no advantage, as has been already
ciently explained, in the substitution of the clinical pi
of asymbolia for that of aphasia. The pathology of dis
ances of the faculty of speech does not deal with *indepen*
diseases. Its object is to describe and analyze the der
ments of the numerous *functions* which come into play in s
ing, writing, and other modes of expressing the thoughts
desires: *theoretically*, to trace them back to their psycholo
anatomical, and physiological causes, and *practically*, to d
mine, partly from the form of the derangement, partly fro

other morbid manifestations that accompany it, the nature and seat of the primary alterations in the organ of language, from which the data for prognosis and treatment can be deduced.

It is true that, at the present time, we are still far from having covered this comprehensive field of study ; we find ourselves only too often obliged to rest content with very indefinite diagnoses, compelled to speak merely of loss of speech or impairment of speech, without being able to define its nature more closely.

This is especially true of many forms of the so-called *functional aphasia*, the inability to speak, which is often observed after intense emotion, in hysteria, and as a symptom of many other neuroses. It is not even always possible to say, whether we have to deal with dysphasic or only with dysarthric derangement; sometimes nothing is certain except the central origin of the impairment of speech.

Loss of the power of speech for a longer or shorter period has repeatedly been known to follow a great *fright*. Dr. Wertner, of Wartberg (Hungary), gave me the details of a case of this sort.

A girl, thirteen years of age, was run over by a wagon. Although she escaped with some slight scratches, she became speechless and remained so for thirteen months. After various methods of treatment had been tried without success, Dr. Wertner at last gave her bromide of potassium. One day, immediately after taking the medicine, she threw herself into her mother's arms and whispered: "Mother, I will speak again." After a few weeks she had entirely regained the use of language.

Sometimes a violent emotion will restore the power of speech, even after it has been lost for a long time.

According to Herodotus (I, 85) the son of Crœsus was dumb, but after the capture of Sardes, he saw a Persian attack his father with a drawn sword, and, horrified at the sight, he called out: "Man, do not kill Crœsus!" It is said that he thereafter retained the power of speech to the end of his life.[1]

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[1] *Wiedemeister* (Allg. Zeitschr. f. Psych. 1874. Bd. 28. S. 485) relates the case of a woman, who became speechless as she left the wedding breakfast to start on the wedding trip with her husband, and remained so until she happened to see a church burning. She cried out: "Fire," and at once regained the power of speech.

Hysterical persons frequently lose not only the voice, but also the power of speech, for minutes, hours, weeks, or months.

A cook was subject to sudden attacks of speechlessness, which came on sometimes several times a day, and would last for hours. During the attacks she was unable to utter a word. All sorts of treatment were tried, without success. Finally she was directed to say under her breath at the commencement of every attack, the words: "God the Father, Son and Holy Ghost," then to turn herself around and to spit out, repeating at the same time inaudibly the words, "That is for thee," while thinking of the devil. This remedy invariably stopped the attack at once.

A lady, somewhat over thirty years of age, suffered from hysteria, with paralysis of the legs and of the left arm. At intervals she lost the power of speech for hours or days at a time, but pressure on any spot in the lateral regions of the neck always relieved this "aphasia" immediately. On removing from my neighborhood she became entirely mute, and for a whole year communicated with her attendants only by writing. Finally the right arm also was affected with paralysis, but she still retained sufficient power to point successively with a little wand to the letters of a large alphabet, by which means she made known her wishes and answered questions. Pressure on the neck was no longer of any use. I had the patient, who had a great regard for me, brought to my hospital, where I gave her a pleasant room and ordered a *placebo*, at the same time stating in her hearing that I confidently expected great results from it. In eight days she was able to speak as well as ever, and could again use the right hand for drawing, writing, and feminine handiwork. Six months later she had to return to her home. The journey proved very harassing to her, a paralysis of the bladder set in, the right arm became permanently paralyzed, and a few months later she died with all the symptoms of a diffuse myelitis.

The word-pictures in such cases are intact, as are also the connecting links between thoughts and words. Have we to deal here with a hiatus somewhere between the picture and the muscles? Or can it be that the combined cortical and infra-cortical excitations, by which the idea is converted into the spoken word, are too weak, and that the "traitement moral" acts by strengthening these excitatory phenomena by means of the psychical stimulation?

After *epileptic attacks* there is often loss of speech for hours, days, or weeks at a time.[1] Ogle[2] saw complete inability to speak set in in the course of *chorea*, and Hughlings Jackson[3] saw the same after *hemispasmus* of the right side. In *catalepsy* the power of speech is lost during the attacks, and the same thing may happen in *ecstasy*.

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[1] Several such observations have been already reported. Another has been published by *Gooday* (Med. Times and Gaz. 1864, Dec.).

[2] *A. Clarus*, Ueber Aphasie bei Kindern. Jahrb. f. Kinderheilkunde. 1874. Bd. VII. S. 369 und S. 396.

[3] Ibidem.

It is almost useless to enumerate all the diseases, in connection with which the so-called aphasic disturbances have been observed. We have already mentioned the fact, that very trifling causes are often sufficient to interfere with the function of speech, and even to abolish it for a time. It is not surprising, then, that *the most diverse severe general diseases, particularly such as are attended by high fever*, should present this complication, especially as they frequently give rise to gross, diffuse and circumscribed lesions of the brain. Thus, aphasia has been observed in *diabetes* (Trousseau), in *morbus Brightii* (Trousseau, Hughlings Jackson, Baginsky), in *syphilis* (Trousseau, Béhier), in *saturnismus* (Heymann[1]), in *alcoholismus* (Trousseau), in *typhus abdominalis*,[2] in *scarlatina* (Eulenburg, Shepherd, and others), in *measles* (Schepers, Calmeil), in *variola* (Breganze), in *erysipelas* (Trousseau), etc. Cases have even been reported in which *collections of fæces in the large intestine* and *lumbricoid worms* were supposed to have caused the aphasia, since it disappeared when the hardened fæcal masses or the worms were removed by treatment.

It is self-evident that *all possible morbid processes in the brain* can cause aphasia. They may do this in one of two ways: either they involve directly and permanently the conducting fibres and centres of speech, or they impair their functional vigor more mediately and temporarily by pressure, ischæmia, or collateral hyperæmia, or perhaps also by simple reflex irritation and radiation. Thus Schlesinger and Ullmann[3] saw aphasia follow a simple commotio cerebri. It is, moreover, frequently met with as a result of fissures and fractures of the skull, of incised, punctured, and gunshot wounds of the brain, of congestion of the brain, of meningitis simplex and tuberculosa,[1] of hydrocephalus, of abscesses, tumors and entozoa of the brain, and of inflammatory, thrombotic and embolic softening of the viscus.

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[1] Berl. klin. Wochenschr. 1865. Nr. 19-21.

[2] Numerous examples are given by *Clarus*. Strange to say, among the cases so far reported, there are only two "aphasic" girls to ten "aphasic" boys. In eleven of the cases there was no paralysis; in one, that was observed in the Julius Hospital at Würzburg (*Gerhardt*), there was hemiplegia of the right side, probably of embolic origin.

[3] Wiener med. Presse. 1869. No. 41.

Broadbent[2] and Waldenburg[3] have reported very interesting cases of *congenital aphasia* in intelligent boys, and Benedict[4] and Clarus (loc. cit.) have met with similar cases. In all of the four cases the patients were boys. The mother of Waldenburg's patient had been seized with aphasia and hemiplegia of the right side in the third month of her pregnancy. The most interesting of these observations is the one reported by Broadbent, which we will give somewhat in detail.

The patient, who was twelve years of age, understood everything that was said to him, and did what he was told to do, but could not, as a rule, speak any words except "yes," "no," "father," "mother"—the last two words he pronounced like "face" and "moce." He used also an indistinct expression which sounded like "keeger kruger," and which he employed in answer to all questions. Occasionally he uttered a few other words, such as "all right," "thank you." He could write his name, and could copy figures or a few words from a printed card, but was unable, when requested to do so, to write down "yes" or "no," or the name of the street in which he lived. He could not understand a written question, no matter how simple it was; for instance, instead of the "no" that was expected, he wrote "baleve" and "ache," instead of "card," "ke," etc. Strange to say, while he was thus unable to connect the written signs with the spoken sounds, and did not understand written and printed text, he nevertheless understood figures to a certain extent; for instance, he could tell the time by the watch, could write down his age, and do other things of a similar character. His tongue was freely movable and he could repeat after any one, although with some difficulty, the entire alphabet, also ba, be, bi, bo, bu, etc., etc.

In spite of the great mass of recorded facts that no longer admit of denial or suppression, it is as yet impossible to deduce any rule of practical value, for the *prognosis* of the dysphasic disturbances included under the head of aphasia. The curability of the aphasia depends partly on the *nature of the causative lesion*, partly on its *location*. When the primary affection is of a curable nature, such as the weakness due to inanition in convalescence from a febrile disease, or simple cerebral congestion, or hysteria, etc., we may look for a favorable result, even though the loss of the power of speech be complete. On the other hand, in incurable affections, such as tubercular meningitis, new growths in the brain, progressive degenerative processes of the cortex, and the like, there is no prospect of recovery from the impairment of speech, even though it be but slight. In the destructive processes the prognosis is worse, when the lesion is diffuse and progressive, than when it is confined to a circumscribed region. In the latter case it depends on the manner in which the conducting fibres and centres of language are affected; when the injury to these is of a reparable nature—due, for instance, to compression or to collateral fluxion—the prognosis is not as bad as when they are directly and incurably involved. In this last case, again, everything depends on the extent of the part involved, and on the question whether or not the lesion is sufficiently circumscribed, to permit of a compensatory action on the part of other bands of fibres and groups of nerve-cells. Unfortunately, the little that we know about these questions can hardly as yet be reduced to formulas of any practical value. One thing may be safely stated, however, and that is, that the *age* of the patient is an important factor for the compensation of the injury. Children have been known to learn to speak, after astonishingly extensive destruction of the left region of speech, and even of the entire cortex of the left hemisphere, while in old persons remarkably small foci of disease in the cerebral substance sometimes cause permanent aphasia. The *individual docility* also unquestionably exerts some influence in each case.

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[1] In 1872 I had in my wards at Freiburg. a man, forty years of age, who was subject to attacks of ataxic aphasia. In some of the attacks he lost the power of speech completely, but in others he was able to say "ja" (yes); between the attacks bradyphasia existed. After his death we found meningitis tuberculosa with tubercular tumors in the gyrus præcentralis sinister, in the neighboring convolutions, and in the left insula. *Gerhardt* (Jahrbuch f. Kinderheilk. N. F. IX. S. 324) recently reported a case of aphasia in a three year old child, who was suffering from meningitis tuberculosa.

[2] Loc. cit., Case IV.

[3] Berliner klin. Wochenschr. 1873. S. 8.

[4] Wiener med. Presse. 1865. Nr. 49.

It would be a great gain for us if we could deduce the necessary data for the prognosis simply from the *form of the dysphasic impairment*, but here, too, the little we know can be summed up in a few sentences. Sometimes apparently severe attacks of aphasia terminate in recovery (we may refer as an example to the case of Lordat), while in other cases slighter derangements, *ex. gr.*, an amnesia of substantives, remain permanent. As a rule, however, simple amnesic aphasia and the paraphasia which presents the characteristics of absence of mind, seem to vouchsafe the best prognosis, while ataxic aphasia, amnesic aphasia with incoherency or entire loss of the word-pictures, and the choreic form of paraphasia, must be regarded as less promising.

The prospects of recovery diminish in an inverse ratio to the *length of time* the affection persists without sign of improvement. They become worse also when the aphasia is *progressive* and accompanied by paralyses and by an increasing weakness of intellect; in this case life itself is usually threatened.

In the *treatment* of aphasia, the first indication is, if possible, *to remove* the *cause*, such as the weakness from inanition, syphilis, saturnismus, hysteria, cerebral congestion or inflammation, etc. When this indication has been fulfilled, and the impairment of speech itself demands treatment although the morbid process in the brain that occasioned it has disappeared, a *methodical course of instruction* in speaking will be found very valuable, provided always a compensation be still possible. This instruction must, according to the nature of each individual case, be chiefly directed either to the motor co-ordination of sounds into words, or to the memory of words. The co-ordination of sounds will be strengthened by methodical practice in the articulation of letters, syllables, words, and sentences, care being taken not to allow the patient to become fatigued. He must be taught from the first to watch closely the mouth of the teacher. With this, writing and reading lessons may be combined when desired. At first only the words in most common use should be used; they should be repeated again and again until the patient becomes thoroughly familiar with them, and not till then are new ones to be added. By this method we, as well as other observers (Trousseau, Broca, Ramskill,[1] et al.), have succeeded in bringing about improvement and even cure. In amnesic aphasia the patient should be made to repeat daily the missing words, the teacher helping him by repeating each word, or its first syllables or letters, before him. When possible, the words should be committed to memory from a dictionary.

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[1] Med. Times and Gazette. Dec. 27, 1862.

CHAPTER XXXII.

Syllable-stumbling. — Impairment of Speech in Progressive Paralysis with Dementia.

We subjoin now to the dysphasic troubles included by authorities under the collective name of aphasia, the affection to which the name of *syllable-stumbling* has been given. In this affection, the *co-ordination of the word as an articulate unity*, composed of letters and syllables, is affected, not through any defect in the articulation of the sounds and syllables, but in consequence of a *derangement of the processes by which the complete word is developed from them.* The words, as organic entities, become loosened at their joints, and their letters and syllables fall apart and become intermingled, or letters and syllables are inserted in words where they do not belong.

The distortion of the words in *stumbling speech* must be distinguished from that which is due to the defective utterance of sounds, or of *stammering.* In stammering some particular sounds are distorted when uttered separately, but the stumbler articulates every isolated sound perfectly well. It becomes a stumbling-block to him only when he meets it in some word, particularly if it be long and alliterative, or when he tries to speak rapidly. If he speaks slowly, partly spelling the words, he may perhaps succeed, provided the affection be not of a very severe grade, in correctly articulating words that are otherwise impossible for him, such, for instance, as "Constantinopolitanischer Dudelsackpfeifer," or "drei und dreissigste Reiterschwadron." When the articulation of sounds is defective, the subject often succeeds by a great exercise of power in uttering with a sort of explosion, sounds which it is impossible for him to articulate, when he employs only the force required for ordinary conversation.[1] The stumbler, on the other hand, attains his aim the most easily by avoiding all forcible articulation, and spea ing as slowly and quietly as possible. The stammerer, like tl syllable-stumbler, replaces letters and syllables by others th do not belong to the words, but the substitution takes pla in a very different manner. The former inserts only certa definite sounds in place of other definite sounds ; for instanc when there is paralysis of the soft palate, he uses an m, w, j in place of a b or p ;[1] the latter, on the other hand, huddl together, in an arbitrary manner, the letters and syllables of word, or even of several consecutive words, says "Keping instead of "Peking," "goten Murgen," instead of "gut Morgen," and "Artrallerie" or "Rartrillerie" instead "Artillerie."

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[1] I was recently enabled, through the kindness of Dr. Alefeld, of Wiesbaden, to see a man who presented symptoms indicating a cerebral sclerosis, and who was unable to

There is one point, however, at which the stammerer a the stumbler meet. The resemblances of the sounds to o another, and the greater or less difficulty that is met with passing from the position required for one particular sound that required for another, make themselves equally felt in t impairment of speech due to stumbling and in that due to sta mering. It is entirely immaterial whether the transition from o sound to another is impeded in the motor-centre for the co-c dination of words, or further back in the centre for articulati proper. In both cases the impulse, when it passes over o sound, is most readily arrested by kindred letters, and alwa chooses those which present the least difficulties.

For this reason, in cases of syllable-stumbling as well as stammering, we very frequently find a reciprocal interchan between the closely related sounds: ü and i, i and e, œ a e, u and o, hard and soft consonants, etc. Or the prece

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utter a single word in such a manner that I could understand it. His wife was the o one who could understand his lisping. Nevertheless, he repeated for me the ent alphabet, articulating correctly all its elements, but each individual sound was a out, as it were, by a great exertion of strength. In this case, apparently the int hemispheric conduction alone was seriously affected. The acts of swallowing a chewing were not interfered with.

[1] A patient with bulbar paralysis, who was in my wards, pronounced, at a particu period of his affection, p like w, g like j or ch, c like d, and i like e. He pronounc b, d, m, n, a, o, u, correctly ; his e sounded like œ, and he could not pronounce r, l, sch, f and k, at all. The aspirate h could only be articulated when his nose was held

ing or succeeding consonant leads to the use of an incorrect vowel in the middle of a word, because the transition to or from the correct vowel is too difficult. Thus W. Zenker,[1] in a very admirable paper, shows that when a paralytic patient says "Züfall" instead of "Zufall," he employs ü instead of u for two reasons: first, because the jump from z to u is greater than from z to ü, and secondly, because the transition from ü to f is easier than from u to f. The interpolation of vowels also, according to his argument, is accounted for as an effort to facilitate the transition from one consonant to another, *e. g.*, "Schewager" for "Schwager." So also the sounds of the vowels are sometimes prolonged, to gain time to put the organs of speech into the proper position for the articulation of the succeeding consonants.

The co-ordination of the word into a complete and perfect entity formed from letters and syllables, depends on *two conditions:* 1. *The acoustic word-pictures must be correctly put together.* This is no longer the case when the sounds and syllables of the words in a sentence, or of long words, are intermixed. 2. *The sounds must be arranged in correct sequence in the motor-centre for the co-ordination of words.* This is no longer the case, when the patient, whose infra-cortical apparatus of articulation is still normal, interchanges kindred sounds, employing by preference those which are most easily uttered, or interpolates uncalled-for sounds, with a view to facilitate the transition.

Stumbling due to loosening of the connections of the word-pictures, shades off into *paraphasia*, while stumbling due to derangements in the motor-production of the words, shades off into *stammering*. In both directions it is difficult to draw the boundary lines. In the first case we call it paraphasia, when the distorted words still retain a meaning, although it is not the meaning desired by the patient—when, for instance, "Butter" is enunciated instead of "Mutter," and "Kaze" instead of "Kaffee;" stumbling, when instead of "Freiburg," the patient says "Feigfro."[1] In the second case, the imperfection of speech is classed under stumbling and not under stammering, when the patient is able to articulate correctly all isolated sounds without employing unusual force, and when the word, which is uttered at one time incorrectly, can at another time be enunciated properly, especially if success be promoted by attention and a quiet delivery.

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[1] Der willkürlichen Bewegungen Modus and Mechanik in der fortschreitenden Paralysie. Allg. Zeitschr. f. Psychiatrie. 1871. Bd. 27. S. 673.

Great *prognostic* importance is attached to this syllable-stumbling, when it accompanies a commencing mental imbecility. "L'embarras de la parole est un signe mortel," said Esquirol, who, when writing these words, had syllable-stumbling in view. In fact, it is most frequently observed in connection with fatal *progressive paralysis with dementia.* It occurs particularly in those cerebral diseases in which slowly progressing irritative processes, involving scattered points (diffuse dissemination), lead gradually to subversion of the cortical functions and to extensive alterations in the minute structure of the cortex. Exceptionally, however, we also meet with it under favorable auspices. Four years ago a drunkard was brought into our hospital suffering from typhus. During the course of the disease, which was attended by moderate fever, his enunciation became stumbling to a remarkable degree, and continued so as long as the fever lasted. We apprehended a complication of typhus with commencing progressive paralysis, and a psychological expert who saw the patient shared our suspicions. Nevertheless, complete recovery set in.

Syllable-stumbling may make its appearance in the *earliest stage of general progressive paralysis*, at a time when the motility in general does not present the least sign of diminution, and when the movements required for the production of sounds and for all other voluntary objects are still perfect (Parchappe[2]). Long afterwards, too, when the impairment of speech has become permanent and very marked, the patients often still retain power over the movements of the tongue, lips, and cheeks, and perform the operations of mastication, insalivation, and swallowing in a most satisfactory manner (Parchappe). This indicates decidedly an affection of the cortex. It has been confirmed in this particular by the results of the microscopical investigations undertaken by Westphal,[1] who in a number of cases could discover no changes either in the peripheral or in the bulbar portion of the hypoglossal nerve. The value of these negative results is not in any respect lessened by the antagonistic positive results obtained by Lubimoff,[2] who has in a few cases succeeded in demonstrating a proliferation of the connective-tissue cells, and degeneration, with granular destruction and amyloid transformation, of the ganglion cells of the nuclei of the facialis and hypoglossus.

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[1] Compare Chapter XXIX.

[2] Bull. de l'acad. de méd. T. XXX. p. 702.

If we bear in mind the tendency of the disease to extend to all parts of the brain and spinal cord, we will not be surprised to learn that its ravages often involve other provinces of language; that, in addition to the syllable-stumbling, it begets *divers other forms of impairment of speech*, more particularly *actual stammering and stuttering, retarded or precipitate and trembling or bleating*[3] *utterance*, and also numerous *derangements of phonation*, which have been thoroughly studied by W. Zenker. The force of the expiration and the tension of the vocal cords are often permanently and greatly diminished, and the voice becomes weak, monotonous, and lower in pitch. Sometimes the patients become hoarse, or they become unable to regulate properly the tension of the vocal cords and the expenditure of wind. In this last instance, so much air is expended in the production of the first few sounds, that the patient, in order to continue speaking, is compelled to bring his abdominal muscles into play. This can be easily demonstrated by palpation of the epigastrium. "The voice, nevertheless, now sinks to a whisper, and by the time the sentence is finished the patient is completely out of breath."

In view of the extension to *other parts of the cortex*, and of the constantly increasing intensity, of the morbid processes which underlie progressive paralysis, it is easy to understand how other anomalies of a dysphasic nature are gradually superadded to the syllable-stumbling, which often constitutes for a long time the only dysphasic symptom. *Temporary attacks of actual aphasia*, which are sometimes attended by abolition, sometimes only by more or less cloudiness, of the intellectual powers, are not infrequently observed. They may be ascribed to temporary derangements in the circulation of the blood, or, perhaps, to swelling of abnormal, spindle-shaped neuroglia-cells in the cortex, caused by the hyperæmia (Lubimoff). *Permanent* aphasia apparently only occurs in connection with grosser, localized lesions of the cortex. In the case of a young Russian, who was suffering from progressive general paralysis, and had previously passed through a severe attack of syphilis, I observed transient attacks of complete loss of speech, accompanied by signs of cerebral congestion. During the attacks the patient had an imperfect comprehension of what took place about him. Between the attacks, which occurred but seldom, his tongue was slightly tremulous, but freely movable, and he enunciated correctly all sounds and words, with the single exception of the word "blanchissage," which he was unable to co-ordinate; in spite of all his efforts, he invariably pronounced it "blanssichage."

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[1] Archiv für Psychol. Bd. 1. S. 90.

[2] Virchow's Archiv. 1873. Bd. 57. S. 371.

[3] *Duchek* (Prager Vierteljahrschrift. 1851. Bd. 29. S. 32) calls this bleating speech "aegophonic." With regard to the impairments of speech in paralytics, compare also *Brosius*, Allg. Zeitschr. f. Psychiatrie. 1857. Bd.14. S. 37.

These patients finally become subject to manifold *syntactic* and *dyslogical* disorders. They take pleasure in hyperbolic expressions, employ extraordinary, self-created words and inflections, and in grammar and in the formation of sentences fall back to the stage of childhood. The sequence of the sentence is interrupted, because the thread of thought is broken. The words are repeated, in consequence either of psychical weakness or of convulsive haste; in the former case, "Ich bin—ich bin—ich bin," will be repeated, until finally the patient remembers what he wishes to say; in the latter, a rapid "Ich bin—bin—bin—bin" is heard (Zenker).

Localized destructive processes in Broca's region in the frontal lobe do not seem to cause stumbling speech, but rather aphasia as understood by practitioners.

CHAPTER XXXIII.

Dyslogical Derangements of Speech, or Dysphrasias.—Aphrasia Voluntaria, Paranoica, Superstitiosa, and Various other Forms of Dysphrasia.—Hemming and Hawing, Cluttering, Hesitating and Confused Speech.—Obstructive Dysphrasia. Verbal and Thematic Paraphrasia.—Influence of Words, Word-phantasms, and Delusions on Speech.—Perversions of Speech in Idiots, Particularly in Microcephalia.—Influence of Congenital Absence of the Corpus Callosum on the Intelligence.

Under the head of *dyslogical perversions of speech*, or *dysphrasias*, we class those affections of speech which proceed from impairment of the intellect. A certain number of extravagant and incorrect terms of speech that are made use of by imperfectly educated or eccentric individuals, or by persons who are under the influence of certain fixed ideas, or are suffering from weakness of mind or melancholia, constitute a transitional stage between normal speech and morbid dysphrasia proper.

Another form of dyslogia remaining within *physiological limits* is the *dumbness, mutitas sive aphrasia voluntaria*, to which some mentally sound individuals condemn themselves, for the sake of fulfilling religious vows or for other motives, which they keep secret. They retain the power of speech, but they will not use it.

Most tourists who have travelled much in Switzerland during the last fifteen years, will remember having met an old pedler, who, for some unaccountable reason, had condemned himself to absolute dumbness. He carried on his business by means of signs.

Lunatics are often persistently dumb for a long time, even for months or years; they may be thought to be perfectly aphasic, until they unexpectedly begin to speak—*aphrasia paranoica*. It is said, that after being dumb for years, they have begun to speak again shortly before death. The causes of this dumbness are various. In that severe form of melancholia, to which the name of melancholia attonita has been given, the speech often remains for weeks to months entombed in the same numbed condition or immobility that reduces all the other movements to a minimum. The immobility involves both the bodily and the mental powers; all questions are responded to at most by a dumb smile, the meaning of which cannot be unriddled. In cases of insanity, it is religious or other delusions, or frequently also hallucinations, particularly word-phantasms in the form of warning, threatening, or commanding voices, that impose dumbness on the patients. God has commanded through the Holy Scriptures, or the Czar of Russia has decreed by a ukase, or some terrible subjective or objective voice has ordered, the patient to remain dumb for a day, or a month, or forever. Finally, however, some unusual event, some severe bodily illness, or perhaps a new commanding voice, loosens again the shackled tongue.

Next to this complete muteness must be classed the intentional avoidance of particular words, the use of which is held to be forbidden for religious reasons or on the ground of propriety. Many wild tribes, immediately after the death of any person, banish completely and most solicitously from conversation the name of the deceased and all words that might recall him or her to mind; the words thus banished are replaced by other newly-coined expressions. It may seem droll, but it is nevertheless correct, to designate and class this custom as *aphrasia and paraphrasia superstitiosa endemica.*[1]

There are persons who have a bad habit of interpolating inappropriate words in their conversation, either at the beginning or the end, or in the middle of a sentence. This form of dysphrasia, to which Merkel[2] gives the not altogether apposite name of *embololalia*, and which would be more correctly designated by the term *embolophrasia*, is really nothing more than a foolish habit, the cause of which is sometimes to be found in the pleasure that weak-headed or affected individuals take in sonorous words.

Thus J. Frank, in his Præceptis (T. 2. V. 2. Sect. 1. Cap. 2), tells of one of his pupils who always interpolated the words "hedera" and "federa" in his sentences. In making a report, for instance, he said: "The patient has slept well, hedera; had two passages, federa."

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[1] *Tylor* has collected and published a number of examples of this custom (Forschungen über die Urgeschichte der Menschheit. S. 178–189).

[2] Schmidt's Encyklopädie der ges. Medicin. Bd. 6. Art. Stammeln.

A professor in a gymnasium had a habit of foisting the words "wiederum" and "dawiederum" into his sentences, particularly when he desired to be pathetic; when he wished to attain the very acme of pathos, he terminated a sentence with "dawiederumda." On one occasion he astonished his pupils by informing them of the death of a comrade, in the following words: "Der kleine Engländer, der erst einige Tage unserer Klasse angehörte, ist schon in verflossener Nacht *wiederum* gestorben, *dawiederumda* (The little English boy, who lately joined our class, died again last night, dawiederumda)."

Another very pedantic and affected teacher embellished his sentences by inserting the sonorous particles "oe," or "oe-doe," or "oe-doe-woe-doe." He invariably terminated the morning prayer with a fervent "oe-doe-woe-doe" inserted just before the Amen, which he always pronounced Amem!—"Oe-doe-woe-doe—Amem."

This fantastic interpolation of stereotyped words in the conversation is also met with as a result of antecedent cerebral disease.

An old General, during the last years of his life, and especially when excited, continually interrupted his remarks with the expletive "mama." This impediment made its appearance after a *coup-de-soleil.* He said, for example: "This miserable —mama—fellow has expected—mama—other people to—mama—pick his chestnuts out of the fire—mama." Even when he spoke Italian, his sentences were interlarded with this curious "mama." His intellect was not impaired.

Perhaps the case reported by Dieulafoy[1] also belongs to this category. In that case there was, combined with aphasia, a singular prefixion of the word "tout" to all words and sentences enunciated by the patient. It is difficult to decide whether this anomaly should be classed under dysphasia, or under dysphrasia. It reminds us of the "und" (and) with which, in consequence of their lack of skill in the combination of phrases, children are wont to begin their sentences; also of the stereotyped "Also," "Wie gesagt," etc., with which many adult persons initiate their sentences. These, however, are errors due merely to want of practice or to bad habits, while, in the patient in question, there was positive inability to articulate the words or sentences without the prefix "tout." He could say "tout-de-même" and "toujours," but not "même" or "jour" alone. He could not say "vin," but could say "tout le vin," "tous les vins sont bons," "tous les rideaux sont blanchis par la sœur de service," etc.

Some people take pleasure in using the diminutive forms of substantives and even of adjectives, *ex. gr.*, "Das ist gutchen," "kleinchen," and the like. Others give peculiar turns to the terminal syllables of words; as an example we may adduce

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[1] Gaz. des hôpit. 1865. No. 68.

Fritz Reuter's "Durchläuchting" and oth made with a view to attain comic effects. must also mention the so-called "Erbsen" of the children. This is a game in whicl or "erbse" and "erbsen" are appended and are put in place of the final syllables oi of "wir essen Erbsen," for instance, the sen esserbsen Erbserbsen." The one wh rapidly wins the game. Among lunatics eccentricities.

A patient of Westphal's,[1] who was both insane employed the diminutive form of substantives: "Federchen," etc.

Trousseau had a patient, suffering from tempoı syllable "tif" to all monosyllabic words, while o articulated the first syllable, replacing all the others "bontif" instead of "bon" and "bonjour," "ve "montif" instead of "monsieur," and so on.

Persons who become embarrassed and ing, either from bashfulness, or becaus know what to say or how to express the the anomaly of speech which we call her Germans *Gaxen* or *Staxen*, and the Fre anomaly might be christened *angophras* bear some resemblance to the French wor is subject to this defect halts in his spe and interrupts the sentence by drawled o thongs, or nasal sounds: ā or a-a, ē or e ang, etc. Frequently, also, one of thes affixed to the end of a word, and serves to that follows. This anomaly may somet habit, long after all trace of shyness in sp and after the speaker has perhaps atta sureness of diction. In such a case the d lent with regard to its matter, and in a form also, but the occasional interpolatio aeng, and the like, disturbs and distracts the listener. This hesitancy in speech is seldom observed in women. It is sometimes symptomatic of intellectual impairment, and especially of general progressive paralysis (Voisin[1]).

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[1] A. Mau, Ueber aphasie. Diss. Berlin. 1872.—

An analogous, abominable fault is the *hemming* or *hawing* with which inveterate retailers of anecdotes are wont to begin and at intervals to interrupt their recitals, partly in order to call general attention to what is coming, partly with a view to gain time to collect their ideas. This defect originates in the efforts required to clear the throat for phonation, when catarrh is present, but after a time the hawking is continued, although there may be no mucus in the larynx. In very many cases the respected speaker would please his listeners better if he would expectorate his sputum, and keep his recollections of "Meidinger" for his own private edification.

The *improper repetition of words, phrases*, and *entire sentences* presents some analogy to hemming and hawing. When the repetition is made intentionally, with a view to accentuate certain words or thoughts, and to impress them on the mind of the hearers, it is of course not incorrect. Sometimes, however, it is the result of a bad habit, or of an uncertainty in the process of thinking or of speaking. It very frequently accompanies dysphasic conditions. Morel[2] has met with it as a symptom of hypochondriacal depression.

A lady, actuated by the fear of losing her speech, often repeated the same word or phrase over and over again for a long time. At other times she would apprehend a paralysis, and to guard against it would keep moving her arm in different directions.

In this case, the motive which led to the persistent repetition of the same word was apparent. In insane persons, however, it is not always possible to discover the reason of the iteration. Frequently the phenomena remind us of a child who, automaton-like, repeats or sings some word or phrase, some rhyme or little verse, until the persons around him can no longer

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[1] Troubles de la parole dans la paralysie générale. Arch. de méd. Janv. 1876. p. 26.

[2] Traité des maladies mentales. Paris, 1860. p. 300. "Voix, parole, perte de la parole."

endure it. Sometimes it is the sound of
meaning, sometimes both, which attra
child, and the word is repeated because
it has a pleasing sound. In insane per
the same thing. An insane man was
the meaningless word "Kitzfleck," whi
over and over again (Brosius). Crazy pe
others with self-created, or sonorous, or f
was in the habit of greeting the physi
"What says Horace?" which he would
less sentences (Brosius).

Many healthy persons have a hab
speaking the last word spoken by their
has its origin in a desire to show that
understand correctly what is said. It
echo, but it must not be confounded wit
of mentally weakened persons, of which
It is also met with in lunatics as a mani
whim.[1]

When the thoughts of a mentally sou
as they do, for instance, when he is op
speech often creeps along with a fati
phrasia. It breaks off suddenly in the
the thread of thought *becomes tangled,*
which show that the drowsy speaker ha
different train of ideas. This phenom
founded with paraphasia, but should
and *paraphrasia.* We find *bradyphr*
developed in a similar manner, in cond
tude and intellectual weakness.

On the other hand, in restless, ex
thoughts flow rapidly and tumultuously
also acquires a precipitate and tumultuo
this natural peculiarity the effects of ne

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[1] Question: "How do you do?" Answer: "How
would you like to do to-day?" Answer: "What
What would you like to do to day? What would you

education are superadded, the defect of speech, called by the Germans *Poltern* or *Brudeln*, by the French *bredouillement*, and by the English *cluttering*, is developed. Writers on stammering and stuttering have designated it by the terms *battarismus* and *tumultus sermonis*, and have with good reason distinguished it from the former defects, which are altogether different in nature. Its development is favored by imperfections of hearing. Clutterers speak too rapidly, do not take time to articulate clearly the sounds and syllables in the words, and clip syllables and even entire words, especially at the end of the sentence; entire sentences even may be entombed in an unintelligible jargon. Alcoholism may cause cluttering in its most severe form. Sometimes it is symptomatic of irritative, morbid processes in the brain.

This defect may easily be confounded with stuttering. In very nervous clutterers the breathing may become oppressed, and in this way a certain resemblance to the other affection be caused; they will then, like stutterers, gasp for breath in the middle of a sentence, and, according to Klencke, may even be attacked with spasm of the glottis. This author states that, at the time when the operative treatment of balbuties was in vogue, a certain operator cut the tongue of an unfortunate clutterer, whom he erroneously thought to be a stutterer, on two different occasions, of course without favorable results. Klencke afterwards effected a cure in this case by means of the didactic method. Eleven other clutterers told him that their physicians had recommended the operation for stuttering, as the only means of treatment affording any prospect of cure.

In general, however, *cluttering may be distinguished from stuttering* by the fact, that it becomes less intense when the patient pays strict attention to what he is saying, while the stutterer succeeds best when he allows himself to speak in an unconstrained manner. The latter acquits himself best in the circle of his friends and relatives, and is at his worst when he tries to speak in public; the infirmity of the former, on the contrary, is most marked when he is in the circle of his intimates, and least noticeable when he is speaking before strangers. Colombat mentions the case of a young preacher who cluttered very badly

when conversing with his relatives and
pulpit delivered his sermons well.

When the cluttering has originated i
education, a cure may be effected by
the cause of his defective speech, exh
speak quietly, and accustoming him
declamations, and methodically cond
think in a measured style, and to place
properly.

A hesitating speech, with longer
that are not, as in hemming and hawin
diphthongs, or nasal sounds, constitutes
logia (*bradyphrasia interrupta*). The
the interruptions, be able to finish what
express his thoughts in correctly constr
not be so fortunate, however, and may ei
or get on the wrong track and beco
sometimes comes to a dead stop in the
description, because he has reached
awakens painful recollections in his hea
to proceed, his words may be smothered
The speech of the victim of melancholia
a similar manner. He sighs and sobs in
or he becomes suddenly silent, and it is
sion of his features that he is no longer
saying. He has given himself up entir
painful feelings. Sometimes the hesita
sion of an intellectual weakness, that
pable of following out his train of th
marked, that he is unable to carry on
natural end.[1] *Hallucinations of hear*
stantly torment the patient, may preve
cluding his remarks. The "voices"
stops, begins again, and so on until, no l
loses all patience, and, careless of wh
breaks out into bitter maledictions of th

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[1] Compare an observation of *Solbrig's*, Allg. Zeitsc

phantasms are sometimes meaningless syllables or corrupted words. A certain patient was greatly irritated, because he thought the boys on the street were constantly calling after him "dex, dex." A minister often heard the people cry out "bi, bi," and from that he deduced the conclusion, that he was about to be raised to a bishopric. An insane woman, at night, heard a voice from the wall, saying: "Auch er errte, er bemeidet dem das Wort." She became very much alarmed at these words, and sought anxiously to find out what these words meant (Snell).

The hesitating speech may become *confused*, although the confused speech sometimes gushes out in a rapid stream. We have already studied the confused speech which is dysphasic in nature, and reaches its acme in choreic paraphasia, but that form which finds its origin in dyslogia, is much more frequently met with. Here the thoughts are confused in consequence of absence of mind or intellectual weakness, and the defect of speech may also develop into a *choreic paraphrasia*. The *distracting influence which the word itself may exert on the thoughts* is a matter of great importance to persons subject to this defect. Every one will be able to recall some occasion on which the casual mention, in the course of conversation, of some name that happened to be the subject of great and general interest, immediately gave a sudden turn to the thoughts of the company, caused the speaker to break off abruptly his unfinished sentence and directed the conversation into an entirely different channel. In diseased conditions, such as mania and dementia, where the association of ideas is destroyed or greatly relaxed, this distracting influence of the word makes itself felt in all its intensity, and may produce such confusion of ideas that the patient is no longer capable of constructing correctly even the simplest sentences. In mania, a senseless jumble of words, connected together only by assonance, alliteration, rhyme, or the like, are whirled out, while in the bombastic utterances of dementia, hundreds of different ideas may be touched upon; an idea is suddenly awakened by a word, a rhyme, or the like, and is just as unceremoniously crowded out in its turn by some other thought.

The *sudden truncation of a sentence* is not always the result of confusion of thought or absence of mind ; it may also be due to a too great fecundity of ideas, which outstrip the words that should express them. Thus, for example, the historian Schlosser, in his lectures at Heidelberg, rushed uninterruptedly onwards, frequently leaving his sentences unfinished. He expected his hearers to complete them in their own minds.

This brings us to a remarkable phenomenon, which can be interpreted as *a loss of the power to put a stop to certain trains of thought, when they have once been set in motion.* For instance, a patient is requested to count up to six, but instead of stopping there, he counts to ten, to a hundred, and even higher, until he is interrupted, or until his voice and perhaps his memory fail him. A patient, who was told to write 2718, was seized with an inordinate fancy for sevens, and wrote 277717. A certain musician, whenever he struck the correct note, invariably tacked on to it a series of notes corresponding to the gamut.

There is another phenomenon which is dependent on *atony of the controlling power of the mind*, and which is met with particularly during periods of fatigue and physical weakness. Some thought, or perhaps some incident connected with the thought, awakens a *converse* idea, which the individual cannot repress, and to which he is forced by an irresistible power to give utterance. In this category, perhaps, belongs the convulsive laugh of nervous women, when exposed to some terrible trial that agitates them violently. Some trivial incident, such as a snore, occurring in the middle of a long, tiresome, sober sermon, will sometimes put an entire congregation into a state of hilarity that many of the members will be utterly unable to control. We might also class here such a case as the following : A sick lady, who was at the same time aphasic, greeted politely a physician called in for consultation, and motioned him to a chair with the most engaging mien in the world, but with the words "cochon, animal." Her son-in-law, who understood what she wished to say, explained to the astonished physician that she meant to express a polite wish for him to be seated (Trousseau). In what other way can we account for the fact that the lady said exactly the opposite of what she felt and thought? Utterances of this sort are usually classed under paraphasia, but it would be more correct to ascribe them to paralogia, and to call them *paraphrasia.*

Finally, we must also bear in mind the *puissant influence* which *predominant political, religious, social, or literary ideas exert on the language of healthy men, and which fixed illusions exercise on the entire speech of persons of unsound mind.* Individuals who, though mentally sound, are wrapped up in particular circles of ideas, and are always ready to discourse on their favorite themes or hobbies, are very liable to digress from the immediate subject of the conversation or lecture, and to find themselves all at once speaking of matters that are entirely foreign to the subject. This is a *thematic paralogia* (*paraphrasia thematica*). Those among the listeners who do not know the speaker sufficiently, and are consequently unable to understand his digression, will be bewildered, and will probably ask themselves: can the man be in his right mind? In monomaniacs the impress of the *delusion* is evident on the voice, accent, carriage, and gestures, on the articulate expressions in words, and on the grammatical and syntactical formation of the sentences. The entire appearance of the patient, as well as his speech, is altered in accordance with the character of the delusion; new words are created, and old ones are rebaptized with new meanings. This is the paralogia of monomania, *paraphrasia vesana.* For instance, they designate by the term "Maurerei treiben," the power which they believe their supposed enemies possess, to pursue them through the thickest walls (Mauer), with their voices, with mockery, taunts, and abuses. Their own ability to hear these voices they define as "Feinhörigkeit," "durch das Hasenohr hören" (quickness of hearing, to hear with the hare's ear), and so on. The word-phantasms they call "Maurerwörter," "Bannwörte," "Bannerwörte," etc.

Brosius had in his asylum a young man who believed himself to be at one time this and at another time that personage. Sometimes he was a famous scholar, and then with head thrown backward, and resting on the back of his chair, he would discourse to the children and nurses, delivering his remarks in a deep chest-voice, with a didactic air and explanative movements of the arms. At other times he was an Italian count or a Norman duke, and with chivalric mien and radiant countenance would boast of his glory and riches. On these occasions he used the Italian or the French language, but interlarded his speech with German sentences. Again, he would be an unfortunate man, neglected by all the world, and would give utterance to his complaints and prayers in a low, whining voice, and with a depressed and cringing air. Sometimes he spoke in a persistent monotone, with but little accentuation and with a moderately pitched voice, sometimes in a passionate style and with a nasal voice, which he thought to be particularly charming.[1]

Before closing this chapter, we must devote a short space to the consideration of the *impairments of speech in idiots.*

The more accurate investigations of recent times seem to support the theory of R. Wagner and Gratiolet, which ascribes *microcephalia*[2] to a cessation in the development of the cerebral hemispheres. The brain remains stationary at a stage corresponding to some particular phase of embryonic life. Even the elementary structure of the cerebrum has been found in a condition corresponding to the earliest stage of embryonic development.[3] If this be correct, we have reason to hope that the results obtained by the study of microcephalia, as well as of other anomalies of the brain due to arrested development, will, when compared with the degree of mental development exhibited by the patients during life, furnish valuable data for the localization of the psychical functions, and of the faculty of speech in particular.

There are microcephali who *cannot learn to speak at all.* The reason for this may lie in the fact, that their mental development has only attained a very rudimentary stage. They do not speak, as Griesinger curtly expresses it, because they *have nothing to say.* There can be no doubt, however, that the reason is often to be found in *an arrested development of the central organs of articulation and speech.* A case reported by L. Meyer[4] furnishes evidence of this.

This idiot could only utter a few scarcely intelligible words, which were rather blown out or chewed out than spoken, and which his relatives construed into "papa," "mamma," and "uncle." Mastication, too, was difficult. On the other hand, the child could express himself well by means of an animated and very intelligible pantomime, and was even able to report on different things that occurred in the asylum.

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[1] *Snell* gives numerous examples of alteration in the manner of speaking and of the formation of new words. See Zeitschr. f. Psychiatrie. 1852. Bd. IX. S. 11.

[2] Compare the very clear and exhaustive exposition of our knowledge on this subject, by *Mierzejeveski*, of St. Petersburg (Verhandlungen der Berliner Gesellschaft für Anthropologie. Berlin, 1872. S. 100.

[3] *Mierjeievesky* et *Boucherau*, Progrès Méd. 1875. p. 702.

[4] Archiv für Psychiatrie. Bd. 5. S. 1.

This view will be all the more readily conceded, in view of the known fact that even intelligent children sometimes remain incapable of learning to speak. The case reported by Broadbent, which is mentioned in Chap. XXXI., is of the greatest importance in this connection.

Other microcephali can only *repeat mechanically*, like parrots, a few words that they have learned by heart, without understanding their meaning and signification.

A third class of these patients learn to *speak to a limited extent, and can express a few wishes.* When excited, they can often utter words that they are unable to articulate under other circumstances. J. Mueller drew attention to this fact. The speech of such idiots is even capable of being perfected by methodical practice; this was demonstrated in the cases of the Aztecs, which Leubuscher reported.

The microcephalus, Elise Schenkel, whom Aeby[1] examined, gave him very accurate information. She had even attended the public school for several years, but was finally obliged to discontinue her attendance, because she was no longer able to follow the exercises. In arithmetic, especially, she could make no progress, and it was with great difficulty that she learned to count up to ten. Singing was her great delight. Her microcephalic brother, Christian, who presented the nimble type, while Elise was only lively, understood simple questions relating to subjects of daily life, and was able to speak a few words correctly.

The aphasic Russian idiot, Mottey, whose case has been so exhaustively described by Mierzejewsky, gave utterance only to the simplest syllables, which were chiefly short, demonstrative, and affectional words: "Hier da, hier da, hier da sie, hier ist est ja, dort, dort;" or "O, o hier da, jenes;" or "O ba, o ba, wessen ist jenes, wessen ist sie, dort, dort," and the like. To all questions addressed to him, he answered: "Hier da, hier da, o, o, hier ist es ja." When pleased, and when any one warmed his hands, he would repeat smilingly: "O ba, o ba, o, hier ist es ja." His brain resembled in shape and in the arrangement of the convolutions, that of a human fœtus at the ninth month, although the conformation of the fossa Sylvii and of the frontal and parietal lobes presented more analogy to the lower stages of uterine life. It surpassed, however, in size and weight the brain of a normal fœtus, and was more developed than that of the anthropoid ape. The idiot was about fifty years of age, and his mental powers were about as developed as those of a one and one-half year old boy, although the brain of the latter would weigh more and its convolutions would be developed. The cerebellum, pons, and medulla were nearly normal in size; but the cerebrum had hardly attained to half the ordinary size. The nuclei of the hypoglossi and vagi were demonstrated microscopically by Prof. Betz.

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[1] Archiv für Anthropologie. 1873. S. 263.

Persons suffering from *acquired idiotism* are sometimes "*everlasting chatterers.*" According to Kind,[1] this seems to be the case under two conditions. In the first place, it is observed in patients who had already attained a certain degree of normal intellectual development, before the idiotism was developed. The words that had been learned are then rolled out mechanically in the most riotous confusion; but they do not give expression to any thought, and are not grouped around any central idea. This last peculiarity serves to distinguish this form of chattering from the *logorrhœa* of lunatics affected with incoherency of ideas, in whom some central idea still glimmers out from the bewildering maze of words. Secondly, it is observed in those in whom the ideas aroused by the impressions made on the senses are so transitory, that they are instantly crowded out by changing impressions with their new ideas and new words. These two conditions may be combined.

In congenital or acquired idiotism, the *faculty for music*, *i.e.*, a good musical ear and a good memory for melodies, sometimes remains, even when speech is impossible. These warblers ("Melodienträller"—melody trillers), as a rule, are incapable of learning anything else (Brandes). According to Zillner,[2] the dumb idiots can be divided into two classes—into those who possess, almost in the normal degree, the power of modulating the voice, and are able to bring forth in succession different, high, clear notes, and those who can only utter a few sharp, harsh sounds. The tension of the vocal muscles exerts the most important influence on the voice; the hoarse, harsh notes are due to their relaxation. In idiots who can speak, we find that it is the formation of the laryngeal sounds that is most defective in some cases, while in others the formation of the lip- and tongue-sounds is more affected.

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[1] In a criticism on the work of *Brandes*, entitled "Der Idiotismus und die Idioten-anstalten," u. s. w. Hanover, 1862. Schmidt's Jahrb. Bd. 115. S. 263.

[2] Med. Jahrb. XI.; Wiener Zeitschr. XXII.

The study of the influence which a *defective formation of special parts of the brain* exerts on the development of the intellect and of the faculty of language, has as yet hardly been begun. Quite recently, Sander[1] and Knox[2] collected and grouped together all the cases of *defective development of the corpus callosum* that could be found in medical literature; the former collected ten cases, the latter fifteen. These cases show that a complete absence or a rudimentary development of the entire commissural system, which binds together the two hemispheres of the cerebrum, entails idiocy. When the corpus callosum alone is wanting, the other commissures being present, idiotism does not always result, but still the patient seems to stop short at a very low stage of intellectual development. Some patients with defective commissural systems learned to speak, although they were idiotic in varying degrees; some of them could only learn a few words, but others were able to answer simple questions, and even learned to read and write. One patient, who could do both these things, was unable to calculate.

CHAPTER XXXIV.

The Two Spasmodic Laloneuroses: Stuttering and Aphthongia.—Nature, Symptoms, Causes, Diagnosis, Prognosis, and Treatment of Stuttering.—Remarks on Aphthongia.

There are two forms of impaired speech that belong in the category of the spasmodic neuroses, viz.: *stuttering* and *aphthongia*. In *stuttering*, *the articulation of the syllables and thereby the entire speech, is impeded in a spasmodic manner.*

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[1] Archiv für Psychiatrie. 1868–69. S. 128.

[2] The London Med. Record. 1875. No. 125.

This impediment does not manifest itself on all occasions, but is apparent only at certain times and under certain circumstances, which, however, are unfortunately very frequently present. *In aphthongia a spasm of the hypoglossus sets in, whenever an attempt to speak is made, in consequence of which speech becomes altogether impossible.*

We will begin with stuttering, which is a very common affection, while aphthongia, on the contrary, is an exceedingly rare disorder.

STUTTERING is a *spasmodic neurosis of co-ordination*, which obstructs the utterance of the syllables, by spastic contractions at the stop-points for vowels and consonants in the articulating tube. This obstruction may be experienced as soon as the patient begins to speak, or it may not be encountered until later on in the middle of his remarks, although all the syllables had previously been articulated with perfect correctness. All isolated sounds are correctly articulated; the affection is a *dysarthria syllabaris*[1] and not a *dysarthria literalis.* In the articulation of a syllable which commences with a consonant, especially an explosive sound, or more rarely of a syllable commencing with a vowel, the speech is suddenly impeded, and the first sound of the syllable or the terminal sound of the preceding one, is repeated usually many times, until finally the obstruction is overcome, and the patient can continue his sentence. This spasmodic obstruction is not experienced at all times; intervals occur during which the stutterer is able to speak without impediment.

If we examine more closely the *processes* that interfere with the proper sequence of the syllables in stuttering, we will find that the three muscular actions, the expiratory, vocalic and consonantal, which are combined in the enunciation of sentences, are not harmonically co-ordinated. The regulating centres in the central nervous system, which preside over the harmonic co-ordination of these muscles in the act of *articulating the sounds*

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[1] Syllaba is derived from συλλαμβανω, I include or combine together, scil. consonants and vowels.

in the syllables, or, as Merkel expresses it, in *the vocalization of the sounds*, are thrown into disorder by even insignificant peripheric, and still more frequently by centric excitations. The irritation caused by a painful tooth, for instance, or most frequently some mental embarrassment, displaces these co-ordinating powers from their too delicate equipoise. The three above-mentioned muscular actions, that take part in the articulate formation of every syllable, are not correctly balanced, with regard either to the force or the duration of the contractions. In consequence of this, the current of air employed in speaking has not sufficient power to overcome the opposing tension of the vocalic or consonantal muscles. On the one hand the respiratory action required for speech is defective, and on the other the tension of the vocalic and consonantal muscles is spasmodically increased; instead of contracting smoothly and for the normal space of time, these muscles are affected with tonic or clonic spasms.

It was not until the third decade of the present century that the differentiation of the two defects of speech, stammering and stuttering, was accurately carried out. The chief credit for this belongs to the Swiss, Schulthess.[1] About the same time, the knowledge of stuttering and stammering was advanced by the researches of Serre d'Alais[2] and Colombat[3] in France, of Arnott[4] and Cormack[5] in England, and of Schmalz[6] in Germany. Among the earlier investigators we must mention Itard,[7] who declared in 1817 that the treatment of stuttering had made no progress in 2,000 years.

Of the later writers on this branch of medicine, we may mention especially

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[1] Das Stammeln und Stottern, u. s. w. Zürich, 1830.

[2] Mémoire sur le bégaiement. Jour. des difformités 1829. No. 2.

[3] Du bégaiement et de tous les autres vices de la parole, etc. Paris, 1830.—Traité médico-chirurgical des maladies de la voix, etc. Paris, 1834.—Orthophonie. 2. Édition. Translated into German by *Fliess*. Quedlinburg and Leipzig. 1840.—Traité de tous les vices de la parole et en particulier bégaiement, etc. Paris, 1843.

[4] *Arnott*, in Elements of Physics or Natural Philosophy, 1830. Chapter "On Articulation," Appendix.

[5] A Treatise on the Cause and Cure of Hesitation of Speech or Stammering, etc. London, 1828.

[6] Ueber Stammeln und Stottern. Clarus und Radius, Beiträge. Bd. 1. H. 4.—Also, Beiträge zu Gehör- und Sprachheilkunde. H. 2. S. 1.

[7] Jour. univ. de méd. 1817. T. VII. P. 129.

Lee,[1] Poëtt,[2] Lichtinger,[3] Klencke,[4] Merkel,[5] Hunt,[6] Rosenthal,[7] Violette,[8] C vin,[9] Wyneken,[10] Coën,[11] and Schrank.[12] Merkel did more than any othe advance the knowledge of the subject, and his account is the one which we in main follow. The physiologists Charles Bell, Marshall Hall, and J. Mueller made stuttering a subject of investigation.—The literature of this impedime speech has reached enormous dimensions; every possessor of an establishmen the treatment of stuttering seems to have been impressed with the idea tha interests of suffering humanity necessitated his appearance as an author. T treatises possess, on the average, about as much value as the current pamphle balneology. Moreover, a number of stuttering physicians and laymen have lished their own experiences, either alone or in connection with those of ot Becquerel, Merkel, Wyneken, and Coën are instances of physicians who stu the defect in their own persons.

With regard to the nomenclature, the ancients seem to have applied the te Hesitatio linguæ seu vocis, *βατταρισμὸς*, *ἰσχνοφωνία* or *ἰσχοφωνία* (Aristotle), cl to *stuttering*, while blæsitas, *τραυλισμὸς* and *τραυλότης*, *ψελλισμὸς* and *ψελλ* were applied to *stammering* in general, and to its special varieties (Schulthess)

According to Merkel, the following *conditions are absolu essential for the articulate formation of the syllables.*

1. *A sufficient supply of air and adequate tension of*

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[1] On Stammering and Squinting, etc. London, 1841.

[2] A Practical Treatise on Nervous Impediments of Speech, Stammering, etc. 5th London, 1842.

[3] Med. Zeitung d. Vereins f. Heilkunde in Preussen. 1844. Nos. 33, 34, 35.

[4] Die Störungen des menschlichen Stimm- und Sprachorgans, u. s. w. Kassel, —Die Heilung des Stotterns. Leipzig, 1862. 2. Aufl.

[5] "Stammeln" und "Stottern" in Schmidt's Encyklopädie der ges. Medicin. I 1844. 2. Aufl.—Anatomie und Physiologie des menschlichen Sprachorgans (Anth phonik). 2. Aufl. Leipzig, 1863.—The same, in Pilz's Zeitschrift Cornelia. Bd. 3. Leipzig und Heidelberg, 1865.—The same, Physiologie der menschlichen Sprache. zig, 1866.

[6] Stammering and Stuttering. London, 1861.—Also, Philosophy of Voice Speech. 1859.

[7] Beitrag zur Theorie und Heilung des Stotterübels. Wien. med. Wochensc 1861. Nos. 35–38.

[8] Études sur la parole et ses défauts, et en particulier du bégaiement. Paris, p. 150 sqq.

[9] Du bégaiement. Paris, 1867.

[10] Henle und Pfeufer's Zeitschr. f. rat. Med. Bd. 31. S. 1.

[11] Anomalien der Sprache. In B. Kraus' Compendium der neueren med. Wi schaften. Wien, 1875. S. 397.

[12] Beitrag zur Lehre des Stotterübels. Allgem. Wien. med. Zeitung, 1875. Nos. 2

expiratory column of air in the air-tubes, and in the accessory tube as far as the point where articulation is accomplished. Without this tension the constriction or interruption of the audible air-column, with which the articulation of the syllable begins, cannot be transmitted with sufficient power to the audible vocal stream.

2. *Subordination of the consonantal to the vocalic muscular action.* The latter must always predominate. This is only possible when the tension in the air-tubes remains approximately the same, until the beginning of a fresh inspiration. During the entire expiratory interval the respiratory muscular system should be kept at the same degree of tension, which should not be unnecessarily weakened by the interpolation between the syllables of uncalled for pauses, during which the store of air is uselessly squandered, or by dwelling too long on the articulation of the consonants.

3. *Preservation of a certain rhythm*, *i.e.*, of the proper sequence of the different acts, allowing to each its appropriate space of time. Those which must be instantaneous should therefore not be allowed to extend over more time than is absolutely necessary. Sometimes one, sometimes another of these fundamental conditions, is more or less *impaired* in cases of stuttering.

1. The apparatus of speech in the mouth and larynx is, as a rule, normal, but very often the development of the thorax and of the respiratory muscular system is defective; stutterers never, however, possess *that degree of control over the respiration which is necessary for speech.* They do not inspire sufficient air, are not sufficiently economical in its expenditure, and allow it to escape without putting it to use; they are sometimes compelled to stop in the middle of a word to draw breath.

In over 600 stutterers, Colombat did not find a single one presenting organic defects of the articulating organs.

Klencke believes that the primary cause of stuttering is invariably *scrofulosis*. This diathesis prepares the soil, and all that is then required for the gradual development of stuttering is the presence of exciting causes. This opinion is not tenable. We examined only recently a man of herculean build, a picture of the most blooming health, who had stuttered from his infancy. Wyneken rejects this theory of Klencke's in the most uncompromising manner.

2. Stutterers *are unable to subordinate the consonantal muscular action to the vocalic*, or to graduate the two at all correctly. In the utterance of the continuous sounds, the stutterer is only occasionally detained longer than is compatible with smoothness of speech. When, however, he tries to vocalize the *explosive sounds*, the characteristic manifestations of his infirmity at once become apparent. He closes the oral canal at one or other of the closing points, according to the nature of the letter to be articulated, and this he does as well as a man who possesses the faculty of speech in perfection, could do it; instead, however, of allowing the vowel to follow without delay, he presses his lips, or his tongue and teeth, or his tongue and palate more firmly together than is necessary, the explosive escape of the air does not take place, the other muscles of the face and those of the glottis, and even the muscles of the neck become spasmodically affected like the muscles of articulation, gesticulatory movements are made,[1] the abdomen is retracted, the head is thrown backward and the larynx is drawn forcibly upward, until finally he works himself into a state of frightful agitation; his heart beats forcibly, his face becomes red and blue, his body is bedewed with perspiration, and he may present the appearance of a complete maniac. Such a *paroxysm of stuttering* may be prolonged until it becomes necessary for the patient to draw breath. The attempts to articulate are then renewed, and continued until at last the desired syllable is more or less correctly enunciated, provided the exhausted patient does not give it up entirely. This description applies only to *extreme cases*. In the *milder forms* of the affection there is a repetition of particular letters and syllables, which greatly impairs the attractiveness of speech, but does not render it unintelligible.

This inability to combine correctly the vocalic and consonantal action is not, however, equally marked at all times. The same person may be able at one time to enunciate long sentences smoothly and easily, while at other times he will stutter frightfully. He is especially apt to do this when he is *confused or nervous*, or when he is *physically exhausted.* Wyneken, for instance, after a night of wakefulness, was unable to utter a word. When conversing with his family or with confidential friends the stutterer speaks well, but when talking to strangers his speech is impeded; he can declaim, sing, argue, and swear without impediment. Wyneken asserts that the supposed rule, that stutterers *can sing* without impediment, is not universally applicable. He himself cannot always say in recitative what he wishes to, although his impediment is very greatly diminished when singing. Schmalz states that stutterers can *whisper* without obstruction, but Wyneken denies this also. He himself cannot say everything in a whisper, but he has not investigated the behavior of others in regard to this point. Perhaps this contradiction of views can be explained by the assumption that stuttering, if severe, is only prevented by whispering when the whisper degenerates into a lisp, and the patient learns to reduce his speech to aphonous utterances. Persons who have undergone the operations of tracheotomy learn after a while to speak in this way. According to Hunt, stutterers *do not stutter when they are asked to do so.* In *solitude* and in *darkness* the speech is, as a rule, unimpeded.

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[1] The extraordinary and often laughable gesticulations and grimaces of stutterers were utilized for the stage by the Italians, even in *J. Frank's* time. There was a standing role for the stutterer (il tartaglia) in their comedies.

3. Stutterers do not succeed in attaining the correct *rhythm* in speaking, because they close too firmly the glottis in the formation of vowels, and the accessory consonantal tube in the formation of explosives, and are consequently unable to relax them when the proper time comes. The rhythm is also impaired by delaying longer than is necessary on the continuous sounds. The controlling power over the rhythmic progress of speech, which the will is accustomed to exert in ordinary conversation, is, however, more frequently impaired by *great mental irritability and nervousness*, than by any other causes. The very fear of stuttering may itself cause the impediment to show itself. When the will is supported by some powerful ally, such, for instance, as the *pathos of a declamation, or the melody of a song, or the feeling of confidence* that animates the patient when he is conversing with his family, or when there is some one at hand to

supply the needed word as soon as the impediment shows itself, the speech is no longer obstructed.

Colombat distinguishes two varieties of stuttering, the *labio-choreic* and the *gutturo-tetanic*.

In the *gutturo-tetanic* form, the closure of the glottis which is required for the utterance of a vowel, is unduly prolonged and develops into a spasm of the glottis. Stutterers affected in this way may find difficulty in enunciating the vowels a, e, i, o, u, even when they occur at the beginning of a syllable. Even persons who suffer usually from no impediment of speech, are sometimes, in consequence of a sudden emotion, of fear or joy, affected with a stoppage of the breath, which causes the initial vowel to stick in the glottis. In the normal mechanism of the utterance of g hard, the glottis becomes entirely closed (Merkel[1]), and this closure also frequently becomes spasmodic. When the stuttering affects the letters k and q, there may be spasm of the glottis combined with a closure of the posterior part of the oral canal, and in the case of k of the nasal cavities also. In this gutturo-tetanic form of stuttering, the mouth remains *open*, and Becquerel[2] named it on that account "*bégaiement ouvert.*"

In the *labio-choreic* form, the utterance of the unfortunate sufferer is arrested by the sounds b, p, d, t, w, m, etc.; he is unable to force the transition to the succeeding vowel. He repeats the consonant three or four times, b b b b, m m m m, and presses the lips forcibly together, or presses the tongue against the teeth, but his efforts are unavailing, until he is forced to stop to draw breath, and thus gains time to quiet down. Becquerel named this form "*bégaiement fermé.*" It is usually accompanied by an active secretion of saliva, which is spirted out when the patient opens his mouth.

In practice, however, it is difficult to make this distinction. Wyneken asserts, that under favorable circumstances every stutterer can enunciate every word without difficulty, while under unfavorable circumstances every word may be obstructed. This may be admitted with the qualification, that the free intervals occur much more rarely in one person than they do in another; the gradations are numerous. The verbal or syllabic combinations at which the utterance is arrested are not the same for different men; combinations that one man can articulate without difficulty, may constitute the principal stumbling-blocks for another person.

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[1] Anthropophonik. S. 853.

[2] Traité du bégaiement. Paris, 1847.

In general, stuttering chiefly occurs in the utterance of the *mute consonants*, the so-called *explosivæ duræ* and *mediæ* (b, p, d, t, g, k), and particularly when they are combined with short vowels or diphthongs (au, ai, eu). It is easier to say "Bahn," "Kam," "Amen," than "Bann," "Kamm," "Ammen" (Wyneken). The *literæ continuæ* (f, ch, l, r, s, etc.), in the utterance of which some air is wasted, and which possess intrinsically a certain ringing quality, and can be prolonged as long as the breath holds out, offer fewer difficulties to the stutterer; they are more easily combined with the succeeding vowels. The transition from the position required for the production of the consonant to that required for the vowel is then not very abrupt. The aspirate h, which is formed in the larynx, and the rolling sound r, offer the least difficulties. The rubbing sounds ch, l, s, j, f, sch, and the resonant n, are rarely obstructed, but the resonant m is more frequently the cause of delay. When the sounds chi, cho, fi, fo, etc., meet with an impediment, the case is regarded as belonging to the severer grades of stuttering.

There are *slighter grades* in which the affection is only revealed by an *unnecessary prolongation of* the letters g, k, w, etc. In our younger days we were more amused than was seemly at the mode of speech of an old nurse, who would say for instance: "K—h—ommen Sie endlich? Der K—h—affee ist schon etw—h—as k—h—alt."

Stuttering is more marked in the morning than in the evening. It is aggravated by fatigue and exhaustion. After a moderate indulgence in alcoholic drinks it abates in intensity, but excessive indulgence aggravates it, and sometimes produces it in healthy persons. A bodily indisposition of any sort is usually attended by an exacerbation of the affection, but in exceptional cases the defect becomes less marked, or even disappears entirely when other diseases set in (Schulthess), or after an injury, a hemorrhage, and the like. In the case of a boy, Wyneken saw the stuttering disappear when an otorrhœa set in, and retur the discharge ceased. A lady stuttered whenever her cat were due. All depressing influences aggravate the defect, moderately exciting influences, which stimulate the fun abate it.

The most reliable computation of the *relative frequ* stuttering will be found in a treatise on this affection by C In France, between the years 1852–62, 6,773 conscript exempted from military service on account of stutterin estimates that there is one stutterer for every 1,000 Fren In Germany it is said to be more frequent than in France.

It is said that stuttering is unknown among the *C* This exemption is probably due to the binding influence strongly rhythmical structure of their language, no less t different intonations being given to one and the same w make it express different meanings. A Frenchman in China, whose mother was a native, stuttered when he French, but expressed himself with the greatest facility Chinese language (Colombat).

Stuttering is very common in some *families*, partly i sequence of *hereditary transmission of the predispo* partly as a result of *bad example and faulty education.* S hess, Colombat, and others, report cases in which stutteri acquired by intercourse with stutterers; in such cases t velopment of the defect is due to imitation, or to a sort o chical contagion. Stuttering is less frequent in women t men. According to Colombat, in 20 stutterers there will males and 2 females. Klencke counted 51 female to 97 mal terers. This comparative immunity of woman is probab to the same causes which make all her movements light more graceful, endow her with finer tact and greater ch manner, and fit her to move in society and to convers readiness, at an earlier age than man.

Stutterers are said to be usually persons of nervou excitable disposition, or of a flighty, sanguine temperame at all events, people of but little power of will (Merkel). eken denies the last statement. For the rest, it is easy to stand the frame of mind that leads stutterers, who, in sequence of their defect of speech, have been ridiculed and kept in the background in society, to withdraw themselves into their homes, where, according to their temperaments, they lead an uncommunicative, distrustful dream-life, or give themselves up to silent frivolities in a fickle, inconstant manner (Klencke).

Stuttering often appears *temporarily* at the periods of dentition and of puberty, or becomes markedly aggravated at these eras. Transitory stuttering is also met with after exhausting mental work and night-watching, after smoking too strong tobacco, during a fit of drunkenness, after epileptic attacks (Wyneken), in hysterical paroxysms (Hasse), in onanists, and as a consequence of indigestion and of intestinal irritation due to worms or to hardened masses of fæces. Transitory and even persistent stuttering has been known to occur as a sequel of acute diseases, such as typhus, whooping-cough, etc. Under all these circumstances, moreover, a previously existing defect may become more intense.

Stuttering may accompany, as a transient or permanent symptom, the *most manifold irritative diseases of the brain and cord*—simple cerebral and spinal irritation as well as actual organic lesions due to traumatism, inflammation, etc. In the chapter on aphasia we described an aphasic form of stuttering, which depended on a circumscribed, cortical encephalitis. Lichtinger differentiates a *spinal* and a *cerebral* stuttering; the spinal form he again divides into *centric* and *excentric or reflex stuttering*. Rosenthal reported a case in which stuttering was one of the first symptoms of tabes. The patient was an officer, and the defect of speech was so marked that he was unable to enunciate even the simplest word of command.

Permanent stuttering probably depends most frequently on *congenital irritability and weakness of the syllabic co-ordinating apparatus;* it is often impossible to discover any exciting cause. It makes its appearance then during childhood, and increases up to the time of puberty; after that era it gradually diminishes, and often disappears spontaneously after middle age. In other cases the patient, by means of long and arduous practice, learns to surmount the defect.

A year ago I was consulted by a young man who stuttered horribly. He had formerly been healthy and strong; he was an excellent swimmer, and had saved several persons from drowning. The last person whom he saved dragged him down to the bottom of the Lake of Zurich, and it was only after efforts which became almost superhuman under the stimulus of mortal fright, that he succeeded in struggling to the shore with his heavy burden. The defect of speech made its appearance immediately after this adventure. He underwent a course of treatment in an establishment for stutterers, but was only slightly improved by it. The affection had already existed for several years at the time I examined him. He was remarkably pale, and his tongue trembled a good deal when protruded; but there were no other morbid symptoms.

It would require too much time and space to examine in detail the numerous *theories* of stuttering that have been advanced by different writers. We will only mention a few of the more prominent ones.

Schulthess compared the stutter-spasm to the convulsive movements of photophobia and hydrophobia, and proposed to call the defect "*phonophobia*" or "*lalophobia*." He admitted consequently the central nature of the affection, which was denied by Malebouche[1] and other contemporanies. These writers attributed stuttering entirely to *a want of skill in manipulating the tongue*, which demanded a purely local treatment.—Colombat believed it to be a "lack of harmony between the nervous excitation which follows the thought, and the muscular movements which should give expression to it." He is evidently a disciple of Rullier,[2] who regarded stuttering as "a disturbance between the idea conceived and the word uttered." Almost all derangements of speech, however, would fall under such a comprehensive category. Schmalz located the essence of the defect in a *primary, spasmodic state of the glottis*.—Lee correctly recognized in stuttering a "*neurosis*, which, like all other neuroses, is *intermittent*. The concomitant organic defects are not the causes, but only conditions which favor the development of stuttering."—Charles Bell regarded stuttering as a *partial chorea;* he admitted consequently the ataxic nature of the neurosis.—Du Soit[3] described it as a neurosis, marked sometimes by tonic, sometimes by choreic spasms of the respiratory organs, by which the action of the will on these organs was impeded.—Lichtinger saw in stuttering a *predominance of the excito-motor over the cerebral system*. This predominance may be brought about in two ways: either the spinal action is normal and the cerebral influence is weakened or abolished, or the cerebral influence is normal and the spinal action is weakened. This view, in which the stutter-spasm is regarded as a *reflex spasm*, was for a time very favorably received by the surgeons, and led to the operative transgressions of Dieffenbach and others.—Merkel attributes stuttering to an *adynamia of the functions of vocalization during speaking*, an adynamia which does not depend on *any anatomically demonstrable, organic defect*, but is located purely and solely in the *psychical sphere*, and especially in the sphere of *volition*, and is dependent on external and physical conditions only in so far as these may influence the mind itself.—Romberg classes stuttering under the head of "*vocal spasms*," but does not attempt to explain the nature of these spasms.—The more recent writers on nervous diseases, such as Rosenthal, Benedikt, and others, agree with us in classing stuttering among the *neuroses of co-ordination*. According to Rosenthal, it is dependent on *congenital weakness* of that portion of the respiratory and vocal apparatus which is located in the medulla oblongata. This apparatus, when severely shaken by some psychical impression in early childhood, never recovers from the shock, but is ever afterwards liable to be excited to uncoördidinated movements by the mere exercise of volition. The morbid influence extends to the processes of neighboring nerve-nuclei, and calls into action the accessory spasmodic movements of the muscles of the face, eyes, tongue, and neck.—Strange to say, Coën takes the very narrow view, that stuttering is merely "the result of a *deficient atmospheric pressure*" *in the lungs*, caused by *disturbances of innervation*.—Wyneken believes that in stuttering, the action of the will on the muscles of speech is fettered by a *feeling of uncertainty*. The stutterer is one who is "*irresolute in speech*."—Schrank defines it to be a *localized nervousness*.

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[1] Précis sur les causes du bégaiement et sur les moyens de le guérir. 1841.

[2] Dictionnaire des sciences méd. Bruxelles. 1828. Art. Bégaiement.

[3] Gaz. med. de Paris. 1840. No. 10.

No man should, at the present day, commit the mistake of confounding stammering and stuttering. The *differential diagnosis* is easy.

1. The individual sounds, as such, present no difficulties to the stutterer, but they do to the stammerer. It is the enunciation of the sounds in their syllabic combinations that the former finds so difficult. Moreover, the stutterer is least likely to be impeded in the articulation of certain continuæ, such as r, l, s, which usually present the greatest difficulties to the stammerer.

2. In stammering, the impeded utterance is not accompanied by the peculiar stutter-spasm.

3. The nervous embarrassment which underlies stuttering, is wanting in uncomplicated cases of stammering. The stammerer usually speaks better, the stutterer worse, when he is questioned about his defect.

4. Rhythm and melody exert an emendatory influence on stuttering, but not on stammering.

5. Stammering is not attended by the peculiar want of proportion between the expenditure of the expiratory force, on the

one hand, and of the phonetic and articulating forces, on the other.

6. Stammering is frequently accompanied by anomalies of the tongue, lips, and articulating organs in general; but malformations, defects, paralyses, etc., are rarely observed in connection with stuttering.

The diagnosis of stuttering *from syllable-stumbling* is also, as a rule, equally easy. In both affections the syllabic utterance suffers in consequence of defects of co-ordination; but in the former the spastic manifestations predominate, in the latter the paralytic; in the former there is simply a dysarthric impairment of the syllabic formation, in the latter there is a combination of dysarthric and dysphasic impairments of the syllabic and word formation; in the former there is a disproportion between respiration, phonation, and articulation, as a result of which the ability to enunciate the sounds in the syllable is suspended, in the latter no such disproportion is noticeable; in the former, speaking is accompanied by a feeling of nervous uncertainty, in the latter it is not; in the former, sounds and syllables are repeated, and syllables cannot be enunciated in consequence of the obstructive spasm, in the latter, sounds and syllables are simply dropped entirely, or are huddled together and interposed in the wrong places.

On the other hand, we must not lose sight of the facts that a stammerer may become subject to stuttering, and *vice versa*, and that stuttering and stumbling may occur together.

Coën[1] has reported a case of *simulated* stuttering.

The *prognosis* depends on the age, temperament, and constitution of the patient, and on the causes, form, degree, and duration of the affection. It is unfavorable when there is a hereditary predisposition to the affection, or when the symptoms indicate great congenital irritability and weakness of the co-ordinating apparatus; when the defect depends on some incurable irritative process in the central nervous system; when the affection is of a severe form and has lasted for a long time, and particularly when the spasms of the glottis are predominant symptoms, or when the spasmodic movements extend to distant groups of muscles; finally, when the patient is advanced in years.

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[1] Allgemeine Wiener med. Zeitung. 1875. No. 48.

Slighter grades often disappear spontaneously during middle age. Klencke cites cases in which the stuttering made its appearance after a long confinement to an insufficient and innutritious diet, and disappeared after the patients had been supplied with adequate nourishment for about a year. According to Rosenthal, young adults, who are obliged to work for the means of existence, and are solicitous about the future, are, *ceteris paribus*, more easily cured of the defect than immature boys and girls. Apparent cures are not unfrequently followed by relapses.

It is said that the severer forms of stuttering, in consequence of the violent exertions required for speech, and the disturbances of the circulation thereby produced, predispose to diseases of the heart, to aneurisms of the aorta and carotids, and to affections of the lungs.

The *prophylactic treatment* of stuttering must be directed against the roots of the evil, against the general debility, and the local weakness of the respiratory organs, and the lack of self-confidence and of power of will. By means of careful nourishment, invigoration of the body by cold sponging, baths, and the like, gymnastic exercises of the lungs, persistent, strict supervision of the mode of using sounds and words, and psychical measures calculated to strengthen the character and brace the sensory and intellectual centres, we will often succeed in counteracting an existing predisposition, and even in curing the affection in its earliest stage. It is, of course, self-evident that the physician must also keep in view the *causal indications*, and endeavor, to the best of his power, to remove the different irritative, morbid conditions of the primæ viæ, generative organs, brain, and spinal cord, which evoke the affection.

The *treatment of the defect itself must be gymnastic and didactic.*

By the *gymnastic* treatment we seek to invigorate the entire system of the patient, and his respiratory organs in particular. This object is attained by means of proper diet, hydrotherapeutics, gymnastic exercises, the Swedish movement cure, and perhaps electricity. Electricity, however, will, at the most, prove serviceable only in strengthening the muscles of the thorax. Rosenthal found that the long-continued application of the induced and galvanic currents to the head, larynx, and hypoglossus, was entirely useless.

The aim of the *didactic* treatment is to re-establish, by means of *pedagogic methods*, the correct co-ordination of the functions of respiration, phonation and articulation. Great credit is due to Colombat, Serre d'Alais and Cormack for the introduction of, and the earliest improvements in, the didactic methods, and to Klencke and others for their present perfection. These methods can be most satisfactorily carried out in special establishments for stutterers, which are under the control of physicians who have made a special study of the affection. The stutterer should enter the family of the physician, who must be at the same time the teacher ; as teacher, he must understand how to win the confidence of his pupil, how to strengthen the control over the will and the emotions, how to impart logical compactness to the thoughts, and how to give force and measured cadence to the movements of articulation.

The treatment begins with exercises in breathing.[1] The stutterer must learn first to fill his lungs completely with air, and then to husband this air, and to expel it slowly and with the proper force. With regard to the details of this and all the subsequent exercises, we must refer the reader to the treatises and works of specialists ; among the German works on this subject, we may mention particularly those of Klencke, Lehweiss,[2] Wyneken, and Coën.

The exercises in breathing are followed by *vocal gymnas-*

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[1] The teacher, Katenkamp, in Delmenhorst, prescribes a period of perfect silence before commencing these exercises. Wyneken's experience in the Delmenhorst establishment led him to regard this preliminary period as one of great value.

[2] Radicale Heilung des Stotterns, etc. Braunschweig, 1868.

tics. The patient must learn to pronounce every vowel both singly and in combination with others. He is made to call out the vowels as loudly and for as long a time as possible, to sing them in tones of different pitch and power, and to pronounce them aloud and in a whisper. Each vowel must be prolonged so that an entire sonorous expiration is expended in its utterance, and must be repeated again and again until the patient is thoroughly convinced that he has mastered it (Wyneken).

Next comes the third stage of the treatment, the *exercises in the combinations of the consonants with the vowels.* They begin with combinations in which the vowel precedes the consonant, then pass to those in which the consonant comes first, next to those which contain several consonants, and so on. In this way, and by dint of daily repetitions and constant attention to the respiration, the patient will gradually be brought to pronounce monosyllables, and will then be advanced to polysyllables, next to simple sentences, and finally to periods. An entire phrase must be spoken like a polysyllabic word, the articulation being subordinated as much as possible to the vocalization. He is then allowed to read aloud, first poetry and then prose, and is at last permitted to pass to free declamation.

Finally, after six to twelve weeks of treatment, the *rhythmic exercises* are begun. The pupil must learn to keep time, to enunciate every phrase very slowly, like a single polysyllabic word, to give to each syllable the same length, and to draw breath wherever there is a stop. After a few weeks he should be introduced among strangers, given commissions to execute, and so on. This regulated speech must be continued for several months at least. Relapses are rather common, and necessitate a new course of treatment. In order to guard against them as far as possible, the convalescent must school himself to keep silent, or to have recourse to rhythmic speech, whenever he finds himself getting excited.

Many attempts have been made to replace this didactic method by *mechanical methods*, and also to combine the two. The ancient writers tell us that Demosthenes spoke with stones under his tongue. Klencke employed for a time a wooden plate, shaped like the body of the lower jaw, which was placed under the tongue. In Paris, in the year 1829, one Schirmann made twenty thousand francs by the sale of small round pieces of wood, as a secret remedy against stuttering. Itard invented a tongue-fork, and Colombat a tongue-bridle. Merkel fastened a clasp made of whalebone on one of the molar teeth of the lower jaw. None of these contrivances ever produced a *permanent* cure. Sometimes, indeed, they proved *hurtful* by the irritation they produced in the mouth, and all of them had at least one inseparable disadvantage in common, viz., they led the patient to regard as of secondary importance that *exercising of the will*, which is really the most important of all the means of treating stuttering. The apparent success that followed their employment was due chiefly to the circumstance, that they forced the stutterer to speak slowly, and consequently in a measured manner.

The genial Dieffenbach[1] committed a great mistake when he proposed to cure stuttering by operative measures, analogous to those employed with success in the treatment of strabismus and club-foot. He made open and subcutaneous transverse cuts through the root of the tongue, and also excised a wedge-shaped piece of the tongue. He had many followers, who emulated one another in inventing all sorts of so-called *stutter operations*. The beneficial effects of these operations persisted only as long as the patients found themselves compelled to put a restraint on the movements of articulation. As soon as the wounds healed, and the pain disappeared, and the tongue consequently again became freely movable, the stuttering invariably reappeared. The history of this sanguinary epoch in the treatment of stuttering will be found in the works of Klencke and Hunt, and in Schmidt's Jahrbücher.[2]

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[1] Die Heilung des Stotterns durch eine neue chirurgische Operation. Ein Sendschreiben an d. Institut von Frankreich. Berlin, 1841.

[2] Bd. 31. S. 136, and Bd. 32. 1841. S. 82.—Froriep divided one of the genio-hyoglossus muscles, and Bonnet cut both of them. Amussat severed the muscular and ligamentous attachments of the tongue to the lower jaw. Philipps divided the hypoglossi. James Yearsley, who believed that narrowing of the fauces was the cause of stuttering, excised hypertrophied tonsils and cut off the uvula.

II. The name *aphthongia or reflex aphasia* was used by Fleury[1] to designate *cramps in the territory of distribution of the hypoglossi, which set in whenever an attempt to speak is made, and render articulate expression impossible.* They bear considerable resemblance to writers' cramp. But few cases of this affection have been published; the first was reported by Dr. Panthel,[2] of Ems, and two others have been recorded by Fleury and Vallin.[3] The spastic excitation of the hypoglossi proceeded most probably from the cerebrum in all of the cases; in two of them the affection made its appearance after periods of great emotional excitement, and in the other it set in, accompanied by severe cerebral symptoms, after an operation on the posterior part of the mouth. This aphthongia, in which the spasm of the hypoglossi that renders speech impossible, is excited by *the intention to speak*, must not be confounded with the disturbances of speech due to lingual cramps dependent on various other spastic neuroses, for instance, chorea.[4]

In Panthel's case, the patient, a peasant boy twelve years of age, was greatly excited by the sudden death of his father. He fainted at the funeral, and remained unconscious for a quarter of an hour. After recovering from the syncope he was sound in body and mind, but for three days he was unable to speak, although he could move his tongue and lips freely, and could swallow without difficulty. When he attempted to speak, his mouth, jaw and tongue remained immovable, but the large laryngeal muscles supplied by the hypoglossi (the sternothyroid, thyro-hyoid and sternohyoid muscles) fell into visible and violent, vibrating movements. The spasms ceased when the efforts to speak were discontinued. Pressure on the muscles relieved the spasms and enabled the patient to speak; this effect persisted as long as the pressure was kept up. Fourteen days after recovery a relapse set in, which was occasioned by fright and lasted two days.

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[1] Gaz. hebdomadaire. 1865. No. 15.

[2] Deutsche Klinik. 1855. No. 40.

[3] Gaz. Hebdom. 1865. No. 17.

[4] See the remarks of *Erb* in Vol. XI., p. 319, of this Cyclopædia. *Hoffmann*, of Suhl, reported a case of severe lingual spasm which rendered speech impossible (Schmidt's Jahrb. Bd. 29. S. 50). The patient was a plethoric man, and a cure was effected by venesection and low diet. *R. Froriep* (Studien zur operativen Heilung des Stotterns. Weimar, 1843) seems to have met with stutterers who were affected with unilateral contracture of the genio-hyo-glossus. He recommended, on that account, the unilateral division of that muscle in the operative treatment of stuttering.

A second relapse of a few hours' duration was brought on by violent emotion a few weeks afterwards.

A child, who was suffering from an inflammatory affection of the neck, was suddenly frightened, and was afterwards seized, whenever it attempted to speak, with a spasm of the lingual muscles that rendered it speechless (Vallin).

In Fleury's case, the patient was a man who had been subjected to the operation of tonsillotomy. The operation was followed by marked disturbances of sensation, loss of taste, aphonia, cerebral congestion, and epileptiform attacks. On every attempt to speak, the tongue became immovably fastened to the hard palate. The intelligence was perfect, and the patient was able to write and to calculate.

CHAPTER XXXV.

Stammering and Lalling.—Dyslalia, Alalia, Mogilalia, Paralalia.—Dyslalia due to Deficient Practice and Defective Education.—Comparison of the Defective Articulation of Sounds in Individuals, Nations, and Races.—Defective Enunciation of Particular Sounds: Rhotacismus, Pararhotacismus, Lambdacismus, etc.—Mechanical Dyslalia.—Hottentotism.—Dyslalia Laryngea, Nasalis Aperta and Clausa, Lingualis, Dentalis and Labialis.

All the disorders of speech which depend upon defective enunciation of the literal sounds have been included under the general head of *stammering* (Stammeln, balbutiement, balbuties, dysarthria literalis). When stammering attains such a grade that the speech is thereby rendered very indistinct or entirely unintelligible, it is called *lalling* (lallatio). This is the term used to characterize the speech of children, before they have learned to pronounce their words so as to be intelligible to all persons—during the period in which their words consist of badly articulated combinations of sounds, that can be understood only by their immediate attendants. When the attendants are silly enough to imitate this lalling, and to communicate with the little creatures in similarly mangled sounds and words, the speech may retain a *childish, lalling character*, which is always unbecoming to an adult, until long after the victims have grown up, and even throughout the whole of life.

Stammering is sometimes *a congenital*, sometimes an *acquired defect;* it is in some cases due to *organic lesions*, and in others is *simply functional in its nature* and *dependent on defective* *education and faulty practice.* The organic lesion is located sometimes in the central nervous system or in the motor nerves of speech, particularly the hypoglossi, and sometimes in the external apparatus of articulation, the tongue, palate, etc.

The manifold causes of *central anarthria literalis* have already been described. We have, in preceding chapters, investigated in an exhaustive manner the morbid processes in the centres of articulation, that lead to disorders in the enunciation of literal sounds. For an account of the derangements of the literal articulation, which are caused by *peripheral injuries of the hypoglossi and faciales*, the reader is referred to the article by Erb, in the eleventh volume of this work, pp. 489 and 523, where he will find descriptions of *glossoplegia* and of unilateral and bilateral *facial paralysis*, of central as well as of peripheral origin. What we have yet to describe are those *two varieties of stammering which originate:* 1. *In defective education and faulty practice;* 2. *In defects of the external apparatus of articulation.* Impediments of the latter variety are classed as *mechanical dyslalia*, and both together constitute the affection to which the term dyslalia in its strictest sense is applied. Entire inability to utter articulate sounds is called *alalia*, in opposition to *mogilalia*, in which only particular sounds are impossible. *Paralalia* is that affection in which the patient, in consequence either of some external mechanical defect or of a bad habit, brings forth a different sound from the one which he wishes to utter.

We will consider first the *dyslalia that depends on defective education and faulty practice.*

Any one may be unable, even though his organs of speech are perfectly developed, to utter certain sounds and classes of sounds which he did not learn during early life. No living man is able to pronounce the speech-sounds of all the nations of the earth. A Lepsius may succeed in expressing them in letters, and a Bruecke may unravel the mechanism of their articulation, but it is beyond the power of even such erudite philologists to articulate them all. In the language of every people we find only certain sounds developed, while others are entirely neglected. We meet consequently with *a national* and *a dialectic mogila-*

lia. The study of these peculiarities is one of absorbing but we can only devote a few paragraphs to them here.[1]

Different languages present the greatest differences gard to their *opulence in sounds.* While the number o nants in Hindostanee has, by the adoption of Sanscrit, Arabian, and Turkish words, been raised to 48, of w originally sprang from the Sanscrit, the English l contains only 20, the Greek only 17, and the Polyne guages have at most 10, and some Australian tongues n than 8.

In certain languages entire classes of sounds are Thus the languages of the Six Nations (the Mohawks, etc.), and that of the Hurons, do not contain the labial v, w, m; they lack consequently the words mamma an which are found in all other languages, and which our learn to speak at a very early age. When an attempt w to teach the Mohawks to pronounce words containing th b, or p, they declared that they would not consent to ma selves ridiculous by closing the mouth in speaking (Tylo languages spoken by the natives of several of the So Islands contain no gutturals. The Society Islanders c pronounce the name of Captain Cook otherwise than What is for these islanders an impossibility, is the delig Shemites. Their languages rejoice in a superfluity of g some of which are unknown among us. The most st sounds that can emanate from the Swiss throat, fall i into the background when compared with the gutturals as it were, vomited up by the Arab.[2]

Sanscrit, which is so affluent in sounds, has no sho o, no soft sibilants and no f. The f is wanting in ma languages—in the Finnish, the Lithuanian, the Mongol others. The d is not employed in the Chinese, the Mexican, and the Peruvian languages. The s is wanting in various Polynesian tongues, and is replaced by a hissing h. The Portuguese discoverer of Brazil noticed that the natives had no f, no l, and no r in their language, and he described them as a people without fé, ley and rey, without religion, laws and king. The Chinese do not use the r. They say ki-li-sse-tu instead of Christus, Ja-me-li-ka instead of America. They replace the r by l, which in its turn is unknown in Zend and in certain Japanese, American, and African dialects. The German guttural ch—as in "ich"—is wanting in French and English, and in return the Germans do not possess the nasal vowels of the Frenchman, and the th and the w of the Englishman. The Russian replaces the German h by g, but takes delight in hissing sounds; he has a soft and a hard sch, a tsch and even a schtsch. The South African races have clicking sounds, and the tribes of the North-west in America have, according to Tylor, chuckling, gurgling, and grunting sounds, which Europeans find it difficult or impossible to pronounce.

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[1] For a more detailed account see *M. Mueller's* Vorlesungen über die W der Sprachen. Bd. II Vorl. 4.

[2] These guttural sounds which are peculiar to the Semitic languages, the "ain," have given rise to many learned discussions. Czermak (Sitzungsber. naturwissensch. Klasse der k. Academie der Wissenschaften. Vol. XXI studied, by means of the laryngoscope, the movements in the larynx that production.

The living languages are not by any means fixed and immutable creations. Like the nations to whom they pertain, they are involved in *a constant process of development or retrogression, which manifests itself in changes in the sounds, words and grammar*. Grimm, in his *law of the progressive transmutation of consonants*, has ferreted out the principle which presided over the consonantal changes, by means of which the words in the languages of the Aryan race were developed from their common roots. After Grimm had broken the ice with regard to the Aryan languages, other philologists attempted successfully to discover the law of orthographical changes in other families of languages. The question that now presents itself is, how can we explain this sound-change? M. Mueller traces it back to the principle of *dialectic growth*. In the original Aryan tongue, the kindred sounds still ran into one another and were not sharply differentiated; it was only in the divergent daughter languages, that these kindred sounds became distinctly separated and defined. In the development of words allied in meaning and root, different nations gave the preference to the classes of sounds that accorded best with their special phonetic organizations; the Hindoos and Greeks chose the aspirates kh, th, and ph, the Goths the mutæ mediæ g, d, b, and the old High Germans the mutæ tenues k, t, p. Even within the limits of these special classes, moreover, we find here one and there another sound preferred in different languages. We find, for instance, the Sanscrit gharma, heat, commencing with the guttural aspirate, while the Greek thermos begins with the dental, and the Latin adjective formus (same as calidus) with the labial aspirate. In support of this hypothesis of dialectic growth, M. Mueller cites the Polynesian dialects. In them, according to Halle, no distinctions are made between b and p, d and t, g and k, l and r, v and w; l, moreover, is frequently sounded like d, and t like k. M. Mueller thinks that the original Aryan tongue existed in just such a condition of *indefinite fixation of sounds*, before the Sanscrit, Greek, Gothic, Sclavonic and Celtic tongues had developed from it.

In addition to the *progressive phonetic development* of languages which we find associated with this dialectic growth, we can also demonstrate an *orthographical decline*. Long words were condensed into short ones, letters and entire syllables being discarded. The French corrupted pater and mater into père and mère, and the English condensed the Anglo-Saxon hlâford and hlœfdige into lord and lady. M. Mueller ascribes this shortening of words to the natural tendency of mankind to choose the least fatiguing way of attaining an object, provided it can be attained as well *with little as with great expenditure of breath and muscular force.* This explanation, despite its simplicity, is probably the correct one for the great majority of the cases. After a time, the concise père and lord were as readily understood as the longer pater and hlaford.

These investigations of the *philologists* are of great value to *pathologists*, in the study of the defects in the production of sounds that are met with in isolated individuals or in entire classes of a people. *For we observe in individuals and in classes of people the same inability to articulate this or that sound, the same disposition to replace it by some other particular sound, the same blending of letters and imperfect differentiation of sounds, and finally, the same tendency, based on* *indolence, to corrupt sounds, syllables and words, that we meet with in entire nations and races.*

One of the most difficult of the letters is the r. Our children, as a rule, do not learn to pronounce r and sch until after they have acquired all the other letters. The Incroyables, who assumed an air of satiety and gave themselves up to a life of womanish idleness, discarded the r completely, and even to-day we frequently see blasé and affected individuals do the same thing. The Incroyables said Bodeaux instead of Bordeaux, just as the Darmstädters say Damstadt, and many Berliners say Keel instead of Kerl, indicating the r only by a slight prolongation of the preceding vowel. Demosthenes, who has been accused of suffering from every possible impediment of articulation, perhaps with a view to utilize him as a brilliant example of phonetic energy for the encouragement of stammerers and stutterers, was, according to Cicero, unable to pronounce the r. It has been proposed to call the inability to articulate the r, *Chinoanismus*, because the Chinese have no r in their language.

The *blending of sounds* may involve the *vowels* as well as the *consonants*. The *blending of vowels* is observed in many persons who pronounce i almost like e, and u almost like o. Schartenmeier, in his edifying song of "Helfer Brehm," rhymes like a thorough Swabian, as the following lines will show:

> "Er studirt das Testament
> Und was sonst für Bücher sind."

He makes Menschen rhyme to wünschen, and Mond to rund, without the slightest hesitation. As instances of the *blending of consonants*, we may mention the case of the Franconian, who pronounces the words "Brod backen" in such a way that we are unable to determine whether he says "backen" or "packen;" also the case of the child, who pronounces the word "komm" half as "komm" and half as "t-h-omm."

Schmalz proposed to *designate the interchange of vowels and diphthongs* by the term "*Phoneentallaxis*," and the *interchange of consonants* by the term "*Symphonallaxis*." These monstrous words may perhaps be made useful in measuring the amount of syllabic co-ordinating power possessed by literati, who are suspected of syllable-stumbling.—Most of th[illegible] defects belong in this category. Many are turned to a[illegible] in the production of comic effects. As an example, we [illegible] to one of the stammering figures of Kladderadatsch. Zwicka[illegible] employs diphthongs instead of vowels—ä instead of [illegible] instead of e—not from laziness, but for a reason that M[illegible] will not allow to be influential in the transmutation of [illegible] languages, viz., the desire to make the sounds more agre[illegible] the ear. What strikes Zwickauer's ear as beautiful [illegible] sounds only comical to the public.

A few of the *defects of articulation in which only* [illegible] *sounds* are affected have been picked out and [illegible] with special names. In doing this, no account was tak[illegible] causes of the defects; it is a matter of indifference whet[illegible] are due to a defective education or to an organic defect.

1. *Rhotacismus* and *Pararhotacismus*.—The letter [illegible] one whose pronunciation is most frequently defective.

a. The synonyms for *rhotacismus* are Schnarren, [illegible] Lorken and Ratschen in German, Grasseyement or pa[illegible] in French, and rattling or burring in English. There a[illegible] r-sounds: the labial r—an example of which is found in [illegible] which coachmen utter when they wish to make their [illegible] stand—the lingual r and the guttural r. In the Euro[illegible] guages only the two last r-sounds are employed. The [illegible] *guttural for the lingual r is termed rattling or bur*[illegible] many towns and provinces of Germany, rhotacismus is [illegible] universal. The burring may present certain variations, [illegible] in the varying manner in which the posterior part of the [illegible] is applied to the [illegible] of the guttural [illegible] uvula and even the glottis may take part in its production. [illegible] remarks of Brücke and of Merkel on this subject are [illegible] of perusal. Merkel thinks that a short frenulum ma[illegible] [illegible] interfere with the production of the lingual r, an[illegible] burring.

[illegible] the remarks of Förster in the Thesaurus [illegible] [illegible] [illegible] [illegible]—see [illegible] op. cit., p. [illegible] *b*. By *pararhotacismus* we understand the employment of other sounds instead of r.

α. Persons who are laboring under this defect, most frequently set in motion only the sides of the tongue instead of the entire tip, and bring forth an l instead of an r. They pronounce the word "Christus" like the Chinese, and like children say "blaten" instead of "braten," and "gloss" instead of "gross." Alcibiades took pleasure in this form of defective utterance, and used the word "colax," flatterer, when he wished to say "corax," raven.

β. Others do not allow the tongue to vibrate at all, and move only the lips, so that r sounds like w; instead of "gross, braun," they say "gwoss, bwaun."

γ. Many, instead of making the tip of the tongue vibrate, only strike it twice against the superior incisors, and convert the r almost into two t's (Prochaska[1]).

δ. Others, again, endeavor to produce the r-sound in the posterior part of the mouth, but succeed as poorly with the guttural as with the lingual r, and replace it by g or even by the nasoguttural ng. The ng seems to be used particularly in France. The people then say "gagnement" instead of "rarement," and "Figango" instead of "Figaro" (Fournier).

ε. In some, finally, the r is preceded by an unpleasant hissing sound, like s or ds, which is due to the vibrating tip of the tongue being pushed too far forward, perhaps as far as between the teeth. They say, *ex. gr.*, "zraison" instead of "raison" (Fournier).

It is difficult to cure burring when it has existed from childhood. The patient must first be taught to apply the tip of the tongue properly to the teeth and the palate, and to drive a strong stream of air against it, so as to produce a purring vibration of the organ. This purring-sound must then be combined methodically at first with vowels, so as to form syllables and words (Rede, Rabe, etc.), and later with consonants and vowels, as, for instance, in the word "F . . . rrr . . . eiheit." The patient should then be gradually advanced to the most difficult words, *ex. gr.*, "Wrangel," Strumpf (Coën[2]).

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[1] Physiologie, § 221.

[2] *Fournier* has described a peculiar plan of treatment for rhotacism, which was in-

Pararhotacismus is due sometimes to a deficient mobility of the tip of the tongue, dependent on shortness of the *frænum linguæ*. The treatment must then be *surgical* in the commencement, and afterwards *dialectic*. Klencke's opinion, that the defect may also be due to an habitual spasm of the genioglossi, or of other muscles supplied by the hypoglossal nerves,[1] which may necessitate myotomy, has not been corroborated.

For the relief of that form of pararhotacism in which an s or a ds is placed before the r, Colombat recommended a special instrument, called the "*refoule-langue*." This was simply an ivory plate, which was placed under the tongue and fastened to the lower incisors by hooklets.

2. *Lambdacismus* and *paralambdacismus*. Defective pronunciation of l.

There are persons who find as much difficulty in pronouncing the l as the r, and who, instead of it, use d, t, s, j, n or ng (Klencke, Amman). Others, like the Japanese, use r instead of l, and say, for instance, "Horrand" for "Holland." A hunter, who pronounced l like n, was vexed with his terrier because he would not draw a badger; but, instead of the word "schlupfen," to slip into the hole, he used the word "schnupfen," to take snuff. The same man wished to say "Lill's Luise ist verliebt bis in den Hals," and did it with the words, "Ninn's Nuise ist verniebt bis in den Hans."

In some of the Allemannic districts the sharp l after vowels is pronounced like an l *mouillé* or soft j; "Oel," for instance, is pronounced "Oej." In other districts the l is pronounced like ow or uw, thus "Oeòw," instead of "Oel" (Schulthess).

3. *Sigmatismus* and *Parasigmatismus*.—Under this heading are classed the defects that are observed in the pronunciation of S and sch, the S- and sch-stammering.

a. The s may be pronounced with unpleasant sharpness. The French call this "*sesseyement*." It often acquires an unpleasant

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vented by the celebrated *Talma*. *Klencke* states that in his experience it has always proved harmful. *Coën* has described it in detail in another place.

[1] TRANSLATOR'S NOTE.—The author uses the words "genioglossi oder hypoglossi," but as there are no muscles called the hypoglossi, the above seems to be the only way in which they can be translated.

hissing character after the loss of teeth, unless the patient is able, when articulating the letter, to stop up the opening with his tongue, so as to present a properly formed air-canal between the dorsum of the tongue and the incisor-teeth.[1] When he cannot do this, the air is driven through the openings with a hissing noise, and frequently the saliva is at the same time spirted out.

b. Many pronounce the s like the English th. This is effected by pushing the tip of the tongue forward between the teeth, until it touches the under-lip, and at the same time not opening the jaws sufficiently. This constitutes the *lisping* or *blæsitas* of dandies, but it is also met with as a defect which has persisted from the time of childhood.

c. Others alter the s into a rustling sch or a sharp f, by enlarging the air-canal between the lips in the shape of a funnel, instead of narrowing it. They say "Joschef," or "Jofef," instead of "Josef."

d. Others, again, pronounce the aspirated sch or the j of the French like a silent sch, or even both the aspirated and silent sch only like a silent s; they say, for example, "swer" instead of "schwer," just as children, Danes, and many Low Germans do. The Westphalians, as is well known, pronounce sch like sk; instead of "Schinkenschnitte" and "heischen," they say "Skinkensknitte" and "heisken."—Many pronounce z like s.

e. Finally, according to Coën, many persons, when they attempt to utter the sch-sound, bring the tongue into the position for the l-sound, but at the same time expel the air without setting it into vibration, as if they wished to pronounce sch. A rustling, rattling sound is thereby produced, that bears no resemblance either to sch or to s. In Austria, this defect of speech is called "ein Hölzel im Mund haben" (to have a bit of wood in the mouth). The distortion of the speech is still more glaring when such persons expel the air through the nose instead of through the narrowed oral canal. This leads to the production of a sound similar to the softened French gn. Instead of: "Hast du schon die schöne Schwester deines Schülers gesehen?" they would ask: "Hast du gnon die gnöne Gnwester deines Gnülers gesehen?"

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[1] Compare *Merkel*, Physiologie der menschlichen Sprache. S. 186 f.

When this impairment of speech is due to the absence of teeth, its curative treatment requires the intervention of the dentist. When the teeth are perfect, Coën asserts that it is ordinarily only necessary to show the patients the proper position of the tongue, and to make them devote a few days to assiduous practising. Great persistence and patience are only required in combating the defects described under "e." First of all, he makes the patient practise the ch-sound for a few days. To do this, the posterior part of the tongue must be placed in the ch-position, and the sound uttered several times in succession. The patient is then made to pronounce ch and silent s alternately and in rapid succession. Finally, he must learn by practice to hold the tongue in this double position, and to force the air rapidly over the two narrowed points.

4. *Gammacismus* and *Paragammacismus*. Guttural stammering.

There are persons who remain, throughout the whole of life, unable to learn the letters g and k, and who substitute for them d or t. In the case of a Danish nobleman who always substituted t for k, Amman pressed down the tongue with his two fingers, so that it could not be brought into contact with the teeth, and told the patient to pronounce the syllable ka, while the tongue was held in that position. To his great astonishment, the stammerer found himself at once able to pronounce the hitherto impossible letter.

The following case presents a *combination of guttural stammering and stuttering.*

A weak-minded man, who was about forty years of age, was able to pronounce all the letters correctly, except g, k, and x. The g he pronounced, both separate and in words, like d, the k like a sharply aspirated t, and the x like itz. He was occasionally able to say g, but never k. His mouth presented nothing abnormal, except that the velum palatinum hung unusually low. Some few words which contained gutturals were particularly difficult for him, and the attempt to utter them would make him stutter and bring on laryngeal spasm. "Gurgel" and "delikat" are examples of such words; in the former the difficulty began with the first syllable, in the latter with the third. People often made sport of his infirmity. They would give him money to induce him to pronounce the word "Delikatessen-Geschäft," for the sake of the amusement his useless grimaces and gesticulations would afford them. In order to avoid the guttural, he would, like children, leave out the prefix ge before the past participle; instead, for instance, of "Der Kaffe is gut gewesen," he would say "Der Thaffe ist dud wesen."

5. Other letters besides those we have just enumerated may also constitute stumbling-blocks for some persons. Amman tells us, for instance, of a boy who had a very short under-lip and a retreating chin, and was unable in consequence to pronounce the letter f. He made him bring the upper-lip and under-teeth together, and the f was then produced by the first expiration.

II. We turn now to those *impairments of articulation which depend on gross anatomical defects of the external organs of articulation*, viz., to *mechanical dyslalia.*

The organic defects that cause this dyslalia may be *congenital* or *acquired.* Even when the defects are very considerable, intelligent children, provided they are not suffering at the same time from congenital aphasia, make energetic efforts to speak, and imitate, to the best of their ability, the sounds and words used by those about them.[1] It is often, however, impossible for any except their nearest relatives to understand what is meant by the scarcely articulated and unintelligible sounds they utter. This intense form of congenital stammering has been named *Hottentottismus.* This name was adopted in consequence of the groundless belief, that the language of the Hottentots is confined to a few indistinct sounds. This belief was discarded long ago, and its incorrectness has been most clearly demonstrated by the recent and learned investigations of Appleyard (1860) and Bleek (1862).—We will here describe in detail a case of Hottentottism, which was due to a *deformity of the mouth.*

A seven-year-old boy, of sturdy build and intelligent appearance, was brought to the clinic by his relatives. He heard and understood well what was said by those about him, and made great efforts to express his wishes; no one, however, could understand his scarcely articulated sounds and words, except those who had by long familiarity learned to divine his meaning. He repeated all the vowels and consonants from dictation, but most of them were unrecognizable. He articulated the vowels best, but combined them all with h and gave them a nasal intonation; his e sounded like i. All of the consonants b, c, d, t, g sounded like an indistinct, aspirated e or i. Of all the consonants, h was pronounced the most perfectly; f, m, and n also were intelligible. In articulating m, he did not close the mouth completely, but merely laid the lower lip against the upper row of teeth. K and q sounded like h-s-hn, s and x like hŭ-hn.

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[1] The opinion of the earlier writers, of *Amman*, *Schulthess*, and others, that children might be so greatly alarmed by a commencing stuttering, that they would be ever afterwards deterred from any further attempts to speak (Alalia mentalis, *Merkel*), still lacks confirmation. At all events, the children would not be so easily frightened by stammering, as is conclusively shown by the case that is here detailed.

In addition to his faulty articulation, the boy could only chew and swallow very slowly. When he ate soup, some of it would flow back into his plate. The saliva dribbled freely from his mouth. The current from ten elements of the portable galvanic battery of Stöhrer, caused strong gulping movements in the muscles supplied by the hypoglossi.

The boy's head was small and his forehead narrow. The circumference of the head was forty-six ctm. ($18\frac{1}{10}$ in.), the tape being laid over the middle of the frontal bone, the convexities of the parietal bones, and the upper angle of the occipital bone. The greatest transverse diameter of the head was on a level with the ears. The eyes were large, and the interocular space seemed remarkably wide in proportion to the narrow forehead. The nose and mouth presented nothing noticeably wrong in the way of form. He suffered from chronic oral and nasal catarrh. The teeth were sound and white, and had indented edges and slightly striated bodies. The lower teeth projected forward, but nevertheless, when the mouth was closed, lay entirely behind the upper teeth. The anterior part of the tongue was of normal size, but its posterior part was much thicker; it could be projected in a straight line one to two ctm. beyond the under row of teeth, but could not, as was proved by numerous trials, be moved to either side or made to touch the palate. When its tip was pushed upward, a tense frenulum linguæ was found. This was divided. The hard palate was remarkable for the extreme height of its arch; in front, in the region of the foramina incisiva, it formed a deep pit, in which the saliva collected. The soft palate was long and hung low down, resting on the dorsum of the tongue, the uvula pointing backward and to the left.

Amman asserts that he saw a girl in Harlem who was unable to articulate a single letter with the exception of t, and that he taught her to speak correctly in three months.

We will now consider *separately* the evil consequences that defects of the *different parts which make up the external apparatus of articulation* have on the enunciation of sounds. As a preliminary, however, we must devote a short space to the consideration of the office of the *larynx* in the articulation of letters.

A. *Dyslalia laryngea.—The larynx* is essentially the *organ* *of voice*, but it is also an *organ of articulation.* This second office cannot be denied to it, as long as h and the h-sounds in general—such, for instance, as the hha and ain of the Arabs—are retained in the different alphabets. It is a well-known fact, that the h-sound is produced by the air rushing through the widely opened glottis. On the other hand, the old controversy over the question whether the vowels are produced in the larynx or in the mouth, has been decided by Helmholtz in the following manner: the vowels are special tones produced in the oral cavity, when it is put in certain particular positions and blown upon from the trachea, and the vibrations of the chordæ vocales of the larynx serve merely to attune them to different heights. What we call a vowel is consequently the clang-tint of the voice, which results from the form of the sound-waves in the oral cavity that has assumed the shape peculiar to that particular vowel.

Czermak[1] has reported a very instructive observation, which illustrates well what has just been said. In a case of *complete obstruction of the larynx*, which necessitated laryngotomy below the point of closure, the patient could not produce either a loud sound or a whisper. She nevertheless subsequently recovered the ability to speak, although she could, of course, only utter *voiceless sounds*. She spoke in a *whisper*, producing the sounds by means of the air enclosed in the pharyngeal and oral cavities; practice taught her how to condense and to rarefy this air in the most skilful manner. She produced by this means not only consonants, but also vowel-sounds, which possessed very distinctly the peculiar characteristics of the individual vowels. She was, it is true, unable to pronounce the vowels separately, but they were produced in the current of articulatory movements, by the consonantal sounds assuming the specific characters of the vowels. She even learned to replace the laryngeal h by an analogous, feeble, undecided rubbing sound, which she produced in the posterior region of articulation.

The larynx, moreover, as the vocal organ, is of radical importance for the articulation, since the production of the mediæ and of the sounded consonants in general, such as the resonants m, n, ng, is effected with its assistance. In the production of these sounds phonation and articulation are intimately combined. The patient of Czermak could not for this reason differentiate b and p, d and t, g and k. She only succeeded in producing the mediæ, when she managed to force a somewhat stronger stream of air from without inward in the oral cavity, through the occlusion places of the explosivæ. The rubbing sounds l and lingual r, on the other hand, were pronounced distinctly; the resonant m was usually replaced by b, and n by d.

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[1] **Sitzungsber. der mathem.-naturw. Klasse der k. Academie der Wissensch. Bd. XXXV.**

It is to be regretted that Czermak has not reported whether or not he succeeded in this case, in the attempt to supply artificially to the oral organ of speech the two things which were alone wanting, viz., air and voice. He proposed to blow both air and sound into the space behind the tongue, through a properly curved, thin tube, in which a sounding apparatus would be inserted. We will merely mention the fact, that Czerny's ingenious proposal to extirpate a useless or cancerous larynx, and replace it by an artificial one, has recently been put into execution. The patients who have been operated on were really enabled, by the introduction of an artificial larynx, to speak in a high-pitched, monotonous voice, resembling a trumpet in tone.[1] It must be stated, however, that persons from whom the larynx was removed *in toto*, still retained the power to speak intelligibly in a whisper. The artificial larynx merely added voice to the articulated sounds.

B. *Dyslalia nasalis et palatina.*

The defects of articulation that are caused by the *nose* remaining open when it should be closed, and closed when it should be open, may be classed under the general term *dyslalia nasalis*, or, shorter, *rhinolalia*. Two forms may be distinguished, an *aperta* and a *clausa*. This dyslalia nasalis is generally, but not always, a *dyslalia palatina*.

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[1] *Czerny*, Versuche über Kehlkopf-Exstirpation. Wiener med. Wochenschrift 1870. Nr. 27 u. 28.—*Gussenbauer*, Langenbeck's Archiv. Bd. 17. S. 354. Ibidem, 1875. Bd. 4. S. 22.—*Reyher*, Ibidem, Bd. 19. S. 334.—For the case of a cook, who suffered from an obstruction of the larynx, *Stoerk* invented an apparatus that proved adequate for the purpose (Anzeiger der k. k. Gesellsch. der Aerzte in Wien. 1875, Nr. 26). He attached one end of a T-tube to the tracheal canula, and the other to an india-rubber tube, which enclosed a reed-pipe. Rings of hard rubber were inserted in the wall of this second tube. The free end of the T-tube permitted free inspiration, and by forced expiration a sufficient amount of air was made to pass into the mouth.

1. *Rhinolalia aperta*.

For the production of the pure vowels and of all the consonants, with the exception of the resonants m, n and ng, the nasal cavity must be closed by the velum palati. For the production of the resonants, the soft palate must hang down, and leave the passage into the nose open. When this passage is constantly open, the voice becomes muffled and snuffling, and the resonants become excessively prominent, while the explosivæ will be more or less indistinct, in consequence of the diversion into the nose of a portion of the stream of air.

This open rhinolalia, with marked snuffling, is most frequently met with as a consequence of *diphtheritic paralysis*, which is notoriously a bilateral paralysis. It is often also the result of *syphilitic and diphtheritic ulcerations*, and, finally, is not unfrequently due to *congenital or traumatic fissures of the hard and soft palates*. Unilateral paralysis of the soft palate also interferes with the purity of the articulation.

Bruecke[1] studied the impairments of articulation in a person whose *soft palate had been destroyed* by syphilis. The other organs of speech were intact. She articulated the tenues p, t, k, distinctly, but not the mediæ b, d, g, because the narrowing of the glottis, in the phonation which is required for the production of the mediæ, diminished the stream of air, and the subsequent diversion of a portion of the stream through the nose left too little for the production of the explosive sounds at the points of closure in the mouth. The vowels did not have as markedly nasal a character as they would have in the case of a person with uninjured but hanging velum, which would allow the air to pass into the nose.

We are indebted to Langenbeck[2] for some communications on the subject of the defects of speech which accompany cleft palate. He remarks that they have been as yet but little studied, and are of a complicated nature and difficult to investigate.

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[1] Sitzungsber. der k. Academie d. Wissensch. 1858. Bd. 28. S. 63.

[2] Archiv f. klin. Chirurgie. 1863. Bd. 1. S. 1-170. According to *Klencke* (Die Störungen, etc. S. 102), it is principally the letters k and g that suffer in cases of cleft

Persons with cleft palate have often, on account of their disagreeable speech, condemned themselves to complete silence, making use of oral communication only when it was absolutely unavoidable. When the defect has been acquired, the suffering of the patient is often increased by the galling feeling, that he alone is to blame for the curse. When the cleft has been acquired, the speech is more nasal, snorting, and whistling; when congenital, it is predominantly guttural, but it is equally bad and unintelligible in both cases. The articulation of the consonants is more difficult than that of the vowels. The intensity of the deformity of speech depends less on the extent, than on the seat of the defect. Defects in the alveolar process, even when they occasion extensive communication with the nasal cavity, affect the speech least. In cases of congenital defect, the impairment of speech is more considerable when the two halves of the palate are in the horizontal position, than in the rare cases in which they ascend perpendicularly towards one another. Persons who are possessed of an active intellect, learn to regulate the defective formation of the voice and articulation much better than those who remain undeveloped intellectually. In consequence of the extensive space which is placed at the disposal of the tongue in cases of congenital cleft palate, the movements of that organ become excessive, and it rolls about unnecessarily in speaking. It may undergo hypertrophy and attain colossal dimensions. In a case of Fergusson's, it shut off completely the pharyngeal cavity from the choanæ. When the cleft involves the entire jaw, the functions of the lips and teeth are also imperfectly performed.

As a rule, the *operations of uranoplasty* and *staphylorraphy* only partially relieve the defects of speech. These do not usually disappear completely, even when the operation has been performed in childhood, and years have been devoted to methodical

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palate. Ka, ke, ki are always sounded like ha, he, hi; kle and kre like tle and tre. A man with cleft palate addressed Klencke as "Tlenhe." The same author states that when there is an opening in the hard palate, the letters b, p, d, t, s, v, are accompanied by a hissing noise.

practice (Passavant). We must rest contented, when we succeed in bringing about an improvement.

The causes of this want of success are various. The tongue feels shackled in the diminished space at its disposal. The most influential cause, however, is the shortness, hardness, and stiffness of the cicatricial, and often persistently infiltrated, velum palati. These conditions interfere with its movements, and prevent the closure of the nares. Its muscles often take a long time to recover, and to regain the necessary power and ease of contraction. Still, Langenbeck has several times known the speech to become normal in the course of a year, or even less, particularly when the patients were intelligent and practised faithfully. When the defect was acquired, the results were, in general, more favorable.

Passavant thought that better results would be obtained by partially uniting the velum palati to the posterior wall of the pharynx; his expectations, however, have not proved well founded.

Billroth[1] holds that the cause of the failure is to be sought in the defective condition of the uppermost part of the constrictor pharyngis, which, like a sphincter, should facilitate the apposition of the velum palati to the posterior pharyngeal wall.

Within the last few years, American dentists, and after them Préterre in Paris, and Suersen in Berlin, have constructed excellent *obturators*, which often completely remove the defect of speech, especially when it is acquired. This *prothesis* has, to a certain extent, replaced uranoplasty. The obturators, however, cannot always be tolerated. They sometimes produce inflammatory irritation, and even increase the defect. They are, moreover, very dear, and must be renewed from time to time.[2]

A cleft of the uvula alone does not exert any influence on the speech. In amputation of the uvula, the levator should not be shortened, unless there should be a special indication for it, as the singing voice might be thereby impaired (Duncan Gibb[3]).

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[1] Chirurg. Klinik. 1860–67. S. 159.

[2] Schmidt's Jahrb. 1868. Bd. CXLI. S. 135.—Verhandlungen des deutschen Chirurgen-Congresses in Berlin. 1875. S. 34–89.

[3] Lancet, 1872. Feb. 6th.

2. *Rhinolalia clausa.*

When the entrance of air into the choanæ during speaking is prevented by hypertrophied tonsils, adhesion of the velum palati to the pharyngeal wall, polypi, or similar causes, or when the choanæ themselves are obstructed by inflammatory swelling, mucus, polypi, foreign bodies, etc., the so-called *obstructed mouth-tone* is produced. The vocal clang-tints become muffled, and the articulation of the nasal sounds suffers to a varying extent, in proportion to the degree and the location of the obstruction.

Czermak[1] examined a girl fourteen years of age, in whom the *velum palati had become adherent to the posterior pharyngeal wall.* The velum, however, could still be made tense or relaxed, be arched or flattened at will. Strange to say, the girl was able to pronounce sounds that bore a striking resemblance to the resonants—the so-called Purkinje's puffing sounds. She forced as much air as possible through the phonating glottis into the closed oral cavity, and then removed the obstruction with as little sound as possible. She pronounced the vowels *a*, *e*, *o*, *u*, distinctly, and also *i*, when it occurred in the course of speech; alone, it was sounded like a crushed *e*. The *r* was formed by the tip of the tongue.

C. *Dyslalia lingualis.*

1. It is a remarkable fact, that *very extensive congenital and acquired defects* of the tongue may exist without preventing speech, or even without rendering it unintelligible.

Louis,[2] on one occasion, when describing a deformed tongue, which consisted merely of two nodules moved by rudimentary muscles, laid great stress on the fact that the speech is less affected than the mastication and deglutition, in cases of congenital stunting or acquired mutilation of the tongue.

A few years ago a small book appeared which was written by an Englishman, Edw. Twisleton,[3] and was devoted to a detailed investigation of this remarkable fact. The work was written as an answer to the opinion expressed by the learned convert, Dr.

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[1] Sitzungsber. d. Wiener Academie. 1868. No. 8. S. 173–177.

[2] Mem. de l'acad. de chir. Paris, 1774. T. V. p. 486.

[3] The Tongue not Essential to Speech. London, 1873. 8. 232. S.

Newman, that miracles were performed in the post-apostolic period, and happen even at the present day. One of the most notorious of these miracles was the recovery of speech by the so-called African Confessor, whose tongue was cut off at Tipasa, A.D. 484, at the command of the heretical vandal, Huneric. Twisleton shows that it is not necessary to assume a miracle in order to account for the Confessor's recovery of speech. He adduces a number of well-authenticated facts, from both ancient and modern times, which prove beyond possibility of doubt, that *even after as much as two-thirds of the tongue has been cut off, the mutilated person can gradually regain the power of intelligible speech by means of properly directed practice.* A man named Rawlinson, whose tongue Nunneley removed with the écraseur on a line with the anterior pillars of the fauces, was able to speak very intelligibly at the end of six months. He was subjected to a careful examination by Ch. Lyell and Huxley. The only letters he could not pronounce were *d*, *t* and *l*. He was consequently unable to pronounce the name of his native place, Leeds. *T* and *d* sounded like *f*, *p*, *v*, or *sh*; the sounds *r*, *s* and *sch* were also impaired; *k* and *g* at the commencement of words were pronounced well, but at the end of words the *g* sounded like the German *ch* (bich for big). Paget's experience accords with this case. He has seen the power of intelligible speech return after six operations of so-called total extirpation of the tongue. The only sounds that the convalescents could not pronounce were *d*, *t* and *th*, but this did not make their speech at all unintelligible. Otto Weber[1] states, that he has repeatedly found the patients able to speak remarkably well a short time after the operation of extirpation of the tongue, the guttural letters alone being indistinct.

We recognize in this ability to accommodate the tongue to its altered circumstances, and to employ it successfully for the purpose of speaking, even after it has been reduced in size by more than half, one of the greatest of the many miracles of nature that we meet in the mechanism of speech.

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[1] *Pitha* u. *Billroth*, Allg. u. spec. Chirurgie. Bd. III. 1. S. 316. Compare also *Maisonneuve*, Gaz. des Hôp. No. 125. 1853.

2. *Congenital and acquired hypertrophy of the tongue* sometimes renders speech impossible, and sometimes merely causes stammering. *Operative treatment* may prove useful in these cases. Surgical measures are also indicated when the *frenulum is very short and firm*, when the *tongue is fastened by adhesions to the wall of the mouth, when tumors of the tongue, jaw-bones, lips or cheeks are present, when the jaw is fixed by cicatricial contractions, when there is muscular contracture or anchylosis*, and for other mechanical impediments to the free movement of the under-jaw and the tongue.

Klencke states, that dryness of the oral cavity and lips is also a frequent cause of stammering. This can hardly prove detrimental in any other way than by impeding the movements of the tongue and lips. To counteract it he recommends the chewing of the radix pyrethri.

The remarkable facility with which some persons are able to draw *the tongue*, especially after the division of the frenulum, *backward into the pharyngeal cavity, to such a depth that it looks as if it had been shortened by excision*, is worthy of mention here. Impostors have repeatedly made use of this manœuvre to excite pity; they represent themselves as dumb, and state in writing that the tongue has been cut out. One of these impostors, who was examined by Dr. Kieme[1], asserted that his tongue had been cut out by a hostile soldier in the battle of Leipzig. Another, who, as we have been credibly informed, succeeded in deceiving a celebrated anatomist, asserted that he had been mutilated by the hand of an Arab, while serving in the Foreign Legion in Algiers.

D. *Dyslalia dentalis.*

Absence of the teeth and a faulty position of the dental rows prevent the correct articulation particularly of s, sch, f, n, and the English th; t, i, and u also are not clearly enunciated.[2] A properly constructed set of false teeth will correct the impediment caused by loss of the teeth, as soon as the wearer has accustomed himself to its use.

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[1] *Meissner*, Taubstummheit, etc. Leipzig u. Heidelberg, 1856. S. 205.

[2] *Merkel*, Physiologie der menschlichen Sprache. S. 408.

When the teeth are held *too close together*, the vowels and labial consonants suffer. A small clamp to be attached to a convenient tooth, has been recommended as a means of treatment (Krug, Klencke).

E. *Dyslalia labialis.*

Of the labial defects which interfere with the articulation of the letters b, d, p, w, m, and which are amenable to operation, the most important is *hare-lip*.

CHAPTER XXXVI.

Deaf-Mutism and the Education of Deaf-Mutes.

It is not our intention to discuss *deaf-mutism* from an otiatric standpoint. Congenital deafness, or the form acquired during infancy, only interests us in so far as it leads to dumbness.[1]

We will state, first of all, that deaf-mutes *are not necessarily perfectly deaf*, as the word would seem to indicate. We find among them besides persons who have lost entirely the sense of hearing, a few also who are still able to hear and understand loud calling and speaking that is carried on behind their backs. Erhard[2] assures us, that many heard the tuning-fork when placed on the head, and that a deaf and dumb wood-cutter, in whom the conduction through the bones of the head was very perfect, even heard a repeating watch at a distance of 41 inches. Nagel[3] examined 72 deaf-mutes between the ages of 7 and 17 years, and found among them six who could clearly distinguish words spoken loudly into the ear, and whistling and other noises produced behind their backs. In 19 of the cases every trace of the sense of hearing was lost. It is evident, then, that *complete deafness is not necessary for the production of deaf-mutism, and that a high grade of hardness of hearing during childhood*

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[1] The literature of deaf-mutism has been thoroughly compiled up to the year 1855, by *B. L. Meissner*, Taubstummheit und Taubstummenbildung. Leipzig, 1856.

[2] Rationelle Otiatrik. Erlangen, 1859. S. 370.

[3] Monatsschr. f. Ohrenheilkunde. 1868. No. 2.

[illegible] present [illegible] [illegible] the power of speech.

This observation is of practical import[illegible] bear it in mind when seeking for the [illegible] imperfect development of speech in a [illegible] brought by its parents to a physician because it makes no progress in speaking [illegible] that the defective speech cannot be [illegible] because the child hears well. [illegible] may show that although the child [illegible] but imperfectly, and that it [illegible] [illegible].

A very intelligent boy, four years of age, [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] a word. We advised the parents to [illegible] [illegible].

The *causes* of deaf-mutism differ in [illegible] of ordinary deafness, except in [illegible] [illegible] during intra-uterine life or during the time of puberty is *the latest period* [illegible] deprive persons of the command of [illegible] already acquired.

In most of the cases of deaf-mutism this [illegible] is developed during the [illegible] from that period until the [illegible] year [illegible] [illegible] are numerous, and those in which the [illegible] and fourteenth years are [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] before them.

[illegible] [illegible] [illegible] [illegible] whether [illegible] *[illegible] into the world deaf [illegible] become [illegible]* tistics having reference to this point are very contradictory. In Belgium, in the year 1815, the deafness was congenital in 1,376 of the cases, and acquired in 370; in Ireland, in the year 1851, it was congenital in 3,534 cases, and acquired in 419. In Bavaria, in the year 1858, there were 2,362 deaf-mutes, four-fifths of whom had been born deaf; 5 per cent. of the remaining fifth had become deaf after five years of age. On the other hand, between the years 1850–53 in France, 693 children were born deaf, while 1,092 became deaf after birth. In the Berlin asylum, in the year 1871, there were 152 deaf-mutes, of whom 69 had been deaf from birth and 79 had become deaf after birth; of the four remaining cases, nothing definite could be learned.[1]

The dissections of Hyrtl, Bochdalek and other excellent anatomists, prove that *imperfect development of the auditory organs* must be classed among the anatomical causes of congenital deaf-mutism. It is certain, however, that in intra-uterine as well as in extra-uterine life, the causes which most frequently abolish the functions of the auditory nerves, are *inflammatory processes in the cavum tympani and the internal ear.* Several times nothing has been found except *thickening of the ependyma of the fossa rhomboidea.*[2]

The careful examinations which Nagel, Roosa and Beard,[3] and others made of large numbers of deaf-mutes, revealed in the great majority of the cases chronic amygdalitis and pharyngitis, perforation and destruction of the membrana tympani with loss of the hammer and anvil, otorrhœa, growths in the middle ear, or a depressed membrana tympani and great narrowness of the external auditory canal.

Gibb[4] asserts that by means of the laryngoscope he discovered an absence of the vocal cords in two deaf and dumb married people. No one else, however, has met with any analogous cases, although Prinz,[1] Læbus[2] and others have undertaken numerous laryngoscopic examinations with this view. Prinz does not even admit the often quoted opinion of Mansfeld[3] of Brunswick, that the larynx and palate are very frequently found misshapen. He examined, it is true, young deaf-mutes from 8 to 14 years of age, who had undergone a course of instruction, while among those examined by Mansfeld there were older and entirely uninstructed subjects. It is altogether probable that the narrowness and smallness of the larynx and trachea, which Engel[4] demonstrated by careful measurements on the dissected body of a deaf-mute, and the defective development of the velum palati that Mansfeld observed in one-third of all the cases he examined, as well as the thick tongue that he often found, are all attributable to the absence of the articulating movements.

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[1] Compare *Boudin*, Dangers des unions consanguins. II. série. Annal. d'hygiène publ. T. XVII. *Falk*, Zur Statistik der Taubstummen. Arch. f. Psychiatrie. Bd. III. S. 407. Conf. *Fr. Majer*, Henke's Zeitschr. 1864. H. 2 (Statistik der Taubstummen und Blinden in Bayern).

[2] *H. Meyer*, Virchow's Arch. 1858. Bd. II. S. 551. *Voltolini*, Ibidem, 1863. Bd. XXVII. S. 171. *Falk*, loc. cit. S. 418.

[3] American Journal. April, 1867. Canstatt's Jahresb. f. 1867.

[4] Med. Times and Gazette. No. 15, 1862.

In some deaf-mutes we have to deal with a *congenital or acquired idiotism combined with the deaf-mutism.* Both the affections are due to *imperfections in the development of the brain, or to diseases of that organ*, especially affections of an inflammatory nature. Or the idiotism is due to a cerebral affection, which is complicated by a *disease of the internal ear* that caused the deafness.

The deaf-mutes *are not distributed equally over the earth.* In most of the large states of Europe the proportion of deaf-mutes to the inhabitants is a pretty constant one, viz., 1 to 1,580 or 1,590. In some of the smaller states, however, the proportion is much less favorable; in Baden it is 1 to 559, in Switzerland 1 to 503. This is probably dependent on the *cretinismus*, which is *endemic* in Baden and Switzerland. Deaf-mutism is most infrequent in Belgium and Holland, where the proportion is 1 to 2,846. In general, it is *more infrequent in flat than in mountainous countries* (Toynbee), probably only because cretinism is more common in the latter. Of the 2,362 deaf-mutes in Bavaria in the year 1858 (1 : 1,746), the majority were found in the regions where cretinism occurs. In the provinces of Piedmont and Savoy, which contain a great many crétins, the proportion of deaf-mutes in the year 1861 was 1 to 563 inhabitants in the former, and 1 to 443 in the latter; in Hesse Darmstadt, which has many crétins in the Odenwald, the proportion in 1861 was 1 to 829. These exceedingly unfavorable proportions are never met with in regions in which cretinism is not endemic.

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[1] Archiv für Heilkunde. 1858. S. 413.

[2] Canst. Jahresber. 1871. Bd. II. S. 490.

[3] Ammon's Monatsschr. Bd. II. H. 1.—Schmidt's Jahrb. Suppl. Bd. III. S. 311.

[4] Prager Vierteljahrschrift. 1850. 3.

Deaf-mutism is *more frequently* met with in the *country* than in *cities*. In Bavaria the relative proportion of country to city cases was as 128 : 100. In Berlin there is only one deaf-mute for every 2,000 inhabitants.

In the sixth decennium of the present century, *the sum total of the deaf-mutes in all Europe*, with the exception of the Turkish provinces, was 145,000.[1]

The affection is *more common in males than in females*. In Bavaria the excess of male subjects was 18 per cent.

It is a remarkable fact, that the defect is incomparably more frequent among *Israelites* than among Christians. In Nassau, in the year 1864, the proportion of deaf-mutes among the Catholics was 1 to 1,397, among the Protestants 1 to 1,101, and among the Jews 1 to 508; in the department of Cologne, in 1869, the proportion was 1 to 1,814 Catholics, 2,638 Protestants, and 560 Jews (Lent). Of 100 deaf-mutes in Bavaria, 25 belonged to the Catholic, 29 to the Protestant, and 46 to the Jewish religion. Liebreich,[2] who was struck by the frequent occurrence of retinitis pigmentosa among deaf-mutes, found that both the affections were most frequently met with in Jewish children, and that the majority of these children came from consanguineous parents.

The French investigators in particular, and more especially Boudin, have laid great stress on the *consanguinity of the parents*, as a cause of congenital deaf-mutism. Boudin ascribes 28 per cent. of the cases, and English statisticians 25–30 per cent., to this cause. According to Majer, 33 out of 1,000 congenital deaf-mutes in Bavaria are the progeny of marriages between relatives.

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[1] *Helfft* published comprehensive statistics of the deaf-mutes in Europe, in the Deutsche Klinik for 1857.

[2] Deutsche Klinik. 9. Feb., 1861.

Several children in the *same family* are often afflicte deaf-and-dumbness. Out of 1,000 families that containe and dumb members, Majer counted 100 that contained more. Falk tells of a family in which six, and of anot which five of the children were deaf and dumb. He ascril infliction in both cases to alcoholism on the part of the Strange to say, the *intermarriage of deaf-mutes* rarely le *the hereditary transmission* of the infirmity. About one- of the deaf-mutes came from parents who had relatives tha similarly affected (Majer).

The infirmity is met with by far most frequently in the *classes* (Falk).

Meningitis, scarlet fever, typhus, measles, and otitis i are the *acute diseases* that most frequently cause de dumbness during childhood.

Deaf-mutism is often *associated with mental weakness.* sixth of all the deaf-mutes in Bavaria were imbecile: two were possessed of good capacity, and one-third of but Fully 78 per cent. could be taught various employments cording to Schmalz,[1] the intelligent deaf-mute is capable o taught to speak up to the fifteenth year of life.

One of the consequences of deaf-mutism is a *defective d ment of the thorax, the voice, and the organs of articu* The slight development of the thorax predisposes to *pulm consumption*, which very often carries off deaf-mutes soo puberty. Meissner prepared a table of the causes of deat pupils of the Deaf and Dumb Asylum at Leipzig. In 49 of the cause of death was satisfactorily ascertained. No les 32 of them succumbed to pulmonary or laryngeal tubercu all, with one exception, between the ages of 12 and 30 yea

Diagnosis. Impostors betray themselves readily by of orthography when they write, for all deaf-mutes writ perfect correctness and never use provincialisms. Whe cannot write, they may possibly be unmasked by means tuning-fork. Actuated by the fear of betraying them they may deny that they feel the vibrating fork when it is placed between the teeth or held over the vertebræ, and in so doing actually divulge their imposture, since every one feels the vibrations in these situations (Erhard).

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[1] Kurze Geschichte und Statistik der Taubstummen-Anstalten und des Ta men-Unterrichts. Dresden, 1830.

The Education of Deaf-mutes.

Even in the sixteenth and seventeenth centuries, philanthropists of different nations sought by instruction in language to fit deaf-mutes for intellectual intercourse with their fellow-beings, and to help them to attain a happier and more worthy position in society.

The Spanish Benedictine Pedro de Ponce (1570), the English divine John Wallis (1653), the Swiss physician Johann Conrad Amman (1692), who labored for a long time in Amsterdam, and finally the Portuguese Jew Pareira in Rochelle (1745), have won for themselves golden names in the Pantheon of the benefactors of the human race by their efforts in this direction. The Abbé Charles Michel de l'Epée[1] (1771), however, was the first who undertook successfully the difficult task of teaching deaf-mutes to speak. To this task he devoted his entire means and life. His instructions, however, were confined to the written and sign languages, and the efforts of his followers in France, among whom we will only mention the Abbé Sicard, were directed principally to the improvement of the methods of teaching required for this end.

The Saxon schoolmaster, Samuel Heinicke,[2] inspired by Amman's celebrated treatise,[3] solved with wonderful sagacity and energy the most difficult problem in the education of deaf-mutes; he taught them to produce not only signs and written characters, but also articulated, spoken words—he really *made the dumb speak.* His method of instruction was called the *Ger-*

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[1] Instruction des sourds et muets par la voie des signes méthodiques. II. Paris, 1771 and 1774.

[2] *H. E. Stoetzner: Samuel Heinicke*, Sein Leben und Wirken. Leipzig, 1870. *Heinicke* was born in 1729, and died in 1790.

[3] Surdus loquens, seu methodus, qua, qui surdus est, loqui discere possit. Amstelod., 1692, 12. The treatise was translated into German twice.

man, in contradistinction to the *French* method, which is the present day in general use in England. In spite of ous attacks from at home as well as from abroad,[1] Hei method has been generally adopted in Germany, and efforts of his pupils Eschke, Reich, the deaf-mute Teusc others, it has been brought to a high degree of perfection.

It is obvious that the German method has two imme vantages over the French.

1. It is only when the deaf person has mastered the language, that he can be said to be entirely restored world. Until then he is unable to *understand*, and to *ma self in turn intelligible to those who can hear*, unless the first acquired the sign-language.

2. Spoken speech is a *gymnastic exercise for the lun air-passages*; it furthers the development of these orga thereby strengthens the entire constitution.

The instruction is of two kinds: *intellectual* and *mec*

1. The *intellectual* aims at the production and comb of ideas drawn from sensory impressions. This is aim first, by means of pantomimes and drawings, later by m written and spoken words. At first only the simplest w used, but as the pupil progresses, the compound wo gradually and methodically brought into use.

2. The *mechanical instruction* aims at the produc articulated sounds and words. The pupils are taught t closely the *mouth of the speaker as he forms his wor* the visible sound-pictures, and to reproduce them by im The successful imitation of the visible sound-motions i

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[1] De l'Epée attacked the method of Heinicke in his essay: La véritable ma [illegible] les sourds et muets, Paris, 17[illegible].—Heinicke answered with: [illegible] Beobachtungen und Beiträge zur Seelenlehre und zur menschlichen Sprache. Le[illegible] The controversy was resumed in the French Academy in 18[illegible], and was carri[illegible] great vehemence. The [illegible], however, could after all only be brough[illegible] that partially deaf subjects can be taught to speak aloud, notwithstanding th[illegible] the Germans had long before succeeded in teaching the entirely deaf to speak. *[illegible]*, loc. cit., Chap. II.

[2] [illegible] published a useful popular treatise, entitled "Das [illegible] [illegible] aus [illegible] [illegible] [illegible] [illegible] [illegible] 3. Aufl. Dresden, 18[illegible].

tated by studying, by means of palpation, the movements of the thorax and the vibrations of the larynx that accompany the act of speaking. By slow degrees the pupils learn to combine properly the movements of respiration, phonation, and articulation.

The intellectual and mechanical instructions are carried on in this way up to a certain time independently of one another, but the intelligence and speech nevertheless react upon one another, and become intimately interwoven, just as they do when children possessed of the faculty of hearing are learning to speak. For instance, a seven-year-old deaf-mute in Leipzig had learned to write very nicely after six months of instruction, but he was still unable to understand what he had written. He simply copied the written questions that were addressed to him (Meissner). As his instruction proceeded, however, he began to understand the written words.

The readiness of speech which the deaf-mutes attain is more or less perfect, according to their intellectual and mechanical proficiency. It depends also on the degree of deafness, which may be complete or partial, on the more or less perfect formation of the organs of speech, and on the age at which they entered the asylum. Their speech always lacks harmony of sound and accentuation, for the production of which the possession of the faculty of hearing is absolutely essential. It is always monotonous, spelling, hard, barking and too loud. Mansfeld[1] has studied closely the defects which mark the speech of deaf-mutes. They are due partly to the loss of hearing, partly to the defective condition of the organs of phonation and articulation, and naturally vary in accordance with the nature of these lesions. He distinguishes two varieties of defective speech, *paraphonia* and *mogilalia*. 1. *Paraphonia*. The voice is unpleasant, rough, and even hoarse, and sometimes changes suddenly from bass to treble (*p. puberum*); or it is whistling and hissing (*p. sibilans*). 2. *Mogilalia*. The deaf-mute often finds it difficult to pronounce certain sounds, particularly r and k (*traulismus*). He frequently changes the hard into soft consonants, uses s instead of g, c instead of t, d instead of k, l instead of r, etc.

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[1] *Ammon's* Monatsschr. Bd. II. Heft 1.

The written productions of the deaf-mute are remarkable for their correct *orthography*, which may contrast very strikingly with the want of skill shown in the construction of the sentences.

Von Froriep[1] has compiled a short summary of the performances of deaf-mutes, from which we can form an opinion as to the extent to which their *intellectual development* can be pushed by education. He found among them a few painters, engravers on copper and mechanicians, one mathematician, several writers, two poets (Pelissier and Chatelain), and even one musician (the son of General Gazan). The musician wrote a treatise on the production and differences of the tones—a companion to the treatise of the blind Saunderson on colors.

We have already mentioned the fact that many deaf persons, or, to speak more correctly, many persons who are hard of hearing, and others whose bones still conduct sounds, are able to perceive tone-vibrations, and revel in *musical feelings*.

The *sense of touch* sometimes attains an exceptional and almost incredible delicacy in deaf-mutes. Pfingsten,[2] the Director of the Deaf and Dumb Asylum in Schleswig, tells of a deaf and dumb girl who could understand almost everything that was said by watching the mouth of the speaker, and who conversed in the darkness of night with the servant-girl, who slept in the room with her. Pfingsten states that he at first refused to believe this. He became convinced, however, when he found that she could understand what was said by the servant with averted face, provided she were allowed to lay her hand on the bare breast of the speaker. On the other hand, the sensitiveness of deaf-mutes to *painful* impressions seems to be less than that of persons who can hear (Itard).

Combined congenital deafness and blindness is, as a rule, due to arrested development of the brain or to hydrocephalus, and is accompanied by idiocy, paralysis, and incurable dumbness. Children affected in this way usually die early.[1] When the *deafness and blindness are acquired during childhood*, the patient is incapable of learning the spoken language, but can still learn the written and sign languages, as is evident from the history of Laura Bridgeman, which was recounted in Chapter XIV. Great intellectual capacity and a sense of touch which is capable of the most delicate and perfect development, are essential in such a case for successful instruction.[2]—The combination of deaf-mutism with blindness does not seem to be very rare, since out of 2,100 deaf-mutes in Sweden, in the year 1840, 90 were at the same time blind.[3]

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[1] Neue Notizen, etc. Weimar, 1845. Bd. XXXIII. St. 5. S. 72.

[2] *Schmalz*, Kurze Geschichte und Statistik, etc. S. 20.

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[1] Compare *Sichel*, Ann. d'Oculist. LIII. Mai et Juin. 1865.

[2] Compare, Erinnerungen eines Blindgeborenen, nebst Bildungsgeschichte der beiden Taubstummblinden Laura Bridgeman und Eduard Meyster. Nach den französischen und englischen Originalberichten des *P. A. Dufau*, *S. G. Howe*, and *H. Hirzel*, by *J. G. Knie*. Breslau, 1852. 8.

[3] *J. Schmalz*, Ueber die Taubstummen und ihre Bildung. 2. Aufl. Dresden und Leipzig. 1848. S. 511.

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